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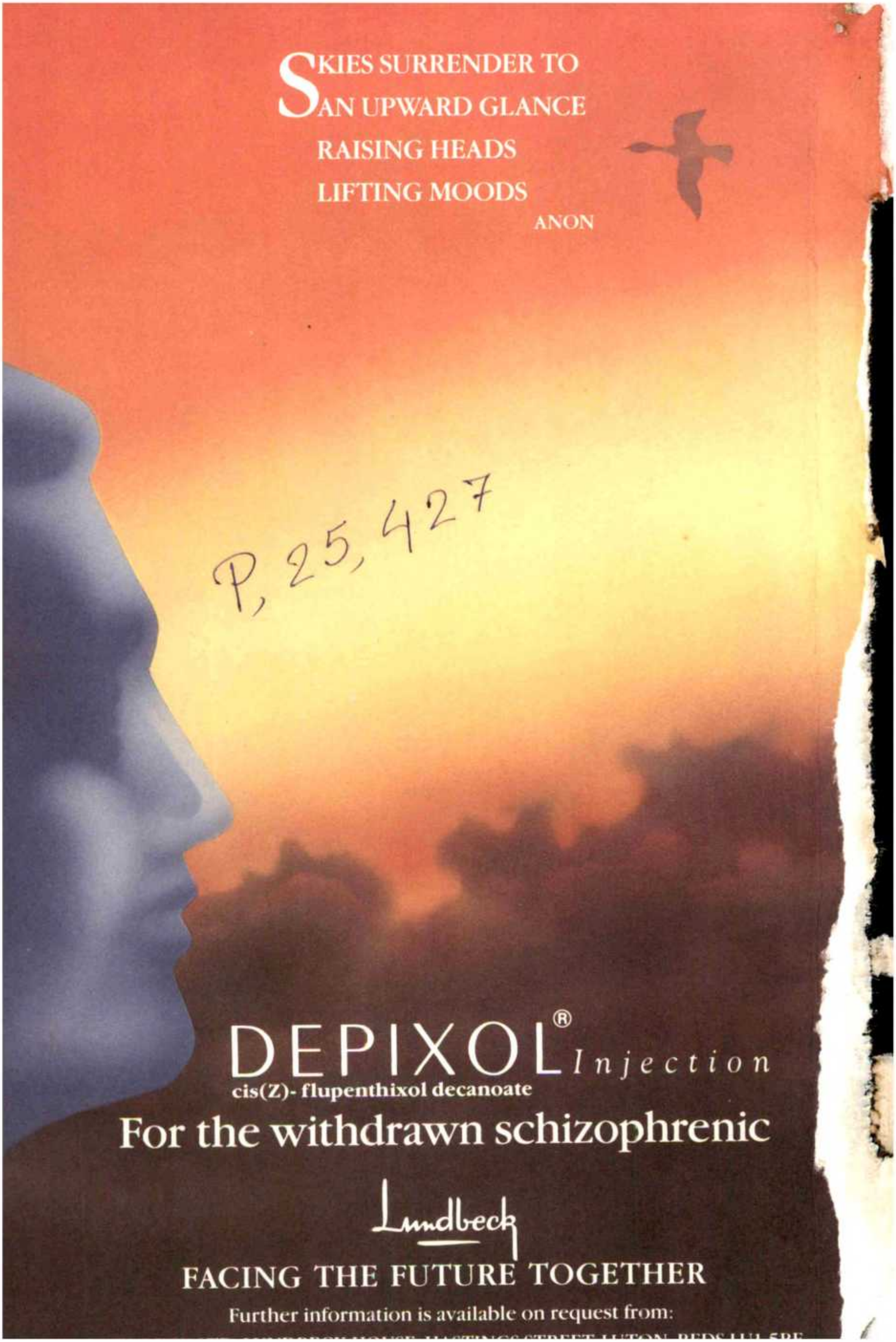
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Review Article

Mental Precursors of Myocardial Infarction

A. APPELS

Both cardiologists and psychiatrists have observed that the onset of myocardial infarction is often preceded by feelings of decreasing energy, general malaise, and minor depression. This paper describes these observations and tries to integrate the findings. It is proposed that the mental state preceding myocardial infarction can be best described as 'vital exhaustion'

The recognition of an impending heart attack is one of the major diagnostic challenges to medicine. Both cardiologists and psychiatrists have therefore investigated the premonitory or prodromal symptoms of myocardial infarction and sudden death. However, few attempts have been made to integrate the observations made by the two disciplines.

Cardiological studies suggest that the premonitory symptoms of myocardial infarction and sudden cardiac death are divisible into three major categories: chest pain, dyspnoea, and fatigue. Estimates of the prevalence of these symptoms are rather crude because it has not yet been established what questions should be asked or what kind of feelings should be grouped into one or more categories. The labels used in most cardiological studies are 'excess fatigue', 'general malaise', and 'lack of energy'. The estimated prevalence ranges from 30% to 60% and is equal to or greater than the prevalence of any other premonitory symptom (Stowers & Short, 1970; Romo, 1973; Alonzo *et al*, 1975; Feinleib *et al*, 1975; Kuller, 1978; Rissanen *et al*, 1978; Fraser, 1978; Klaeboe *et al*, 1987).

Psychiatrists who looked into the mental state of coronary patients observed similar symptoms. Their descriptions contain many similar elements, but they use different terms, such as "hidden withdrawal and masked depression" (Fischer *et al*, 1964), "a combination of depression and arousal affects" (Greene *et al*, 1972), "pseudoneurasthenic syndrome" (Polzien & Walter, 1971), and "emotional drain" (Bruhn *et al*, 1968).

Most cardiologists avoid further interpretation of excess fatigue. Some authors suggest that it is a side-effect of medication or reflects subclinical levels of atherosclerosis. Some psychiatrists are inclined to interpret this state as a breakdown of defence mechanisms before the onset of myocardial infarction. Others explain the depressive symptoms as a reaction to the life-threatening event or as a 'home-going' depression (Billing *et al*, 1980; Hacket, 1985).

Because of the retrosepctive nature of these studies, a number of questions remain unanswered. Which feelings exactly precede myocardial infarction? How long had these feelings existed? What is the most appropriate label for these feelings? What is their origin? Prospective studies of feelings are called for, controlling for classic risk factors. Two such studies are considered; thereafter the remaining questions are discussed.

Quantitative studies of mental precursors to coronary heart disease

The first prospective psychiatric study of the mental precursors to myocardial infarction was published by Crisp *et al* (1984). The database of this study was formed by patients, aged 40–65, who were registered with a group general practice in south-west England. This group was screened in 1969, 1971, and 1973. Nearly all subjects completed the Crown–Crisp Experiential Index (CCEI) on each occasion. During the five-year observation period, 26 men were admitted to hospital with a confirmed diagnosis of severe myocardial infarction. Their scores on the CCEI were compared with those of the remainder of the male study population.

Future coronary patients were found to have significantly more somatic experiences (especially of sweating, heart flutter, and loss of libido) and were significantly more depressed (in particular, feeling sad). Differences between the groups were sought in their scores on the individual items on the CCEI and in a discriminant analysis seeking a linear combination of the variables that discriminate most between individuals destined and those not destined for early infarction. A set of 12 items was found that reflected a state of sadness, coupled with loss of libido and exhaustion. These results should be interpreted cautiously, because change fluctuations may strongly influence the results of a discriminant analysis (Thompson, 1984). No attempt was made to control for confounding factors or for a time-dependent association.

In a new analysis of the same database, Haines *et al* (1987) observed that the scales 'obsessional neurosis', 'depression', and 'hysteria' were predictive of all cases of ischaemic heart disease. However, these scales lost their predictive power when they were controlled for phobic anxiety. This rather surprising result, surprising because an association between phobic anxiety and coronary heart disease had never been mentioned before in the literature, may indicate that only those symptoms of depression, which are also experienced by phobics, are predictive of myocardial infarction.

The second prospective study of the mental precursors of coronary heart disease was conducted by Appels & Mulder (1988). This study was done among 3877 male civil servants of the city of Rotterdam (the Rotterdam Civil Servants Study (RCSS)). The cohort was screened for cardiovascular risk factors between 1979 and 1980 and was followed up for an average of 4.2 years. During the screening, all subjects completed a questionnaire asking about different manifestations of exhaustion, emotional drain, irritation, depression, and sleep problems. This questionnaire, called the Maastricht Questionnaire (MQ), was composed of two parts: form A, consisting of 37 items, developed in an earlier study; and 21 additional questions, derived from clinical interviews. It was hypothesised that future cases of myocardial infarction would have elevated scores on form A of the MQ at screening. Furthermore it was expected that an exploration of the predictive power of the added set of questions would reveal some risk indicators not yet included in form A of the MQ.

Among those free of past or present coronary heart disease at the time of screening, there were 59 subjects who experienced myocardial infarction during the follow-up. Form A of the MQ was found to be significantly associated with an increased risk of future myocardial infarction when controlling for age, smoking, cholesterol, and blood pressure. An analysis of the observed association against length of time interval showed a strong decrease in the relative risk of events occurring during the first, second, third, or fourth year of the follow-up. The age-adjusted relative risk associated with a score above the median of the MQ was found to be 10.05 in the first year of the follow-up, while the risks for the following years were 2.23, 3.04, and 0.68, respectively (relative risk is defined as the ratio of the proportion of respondents experiencing myocardial infarction to that of those who did not). These data indicate that exhaustion is a strong short-term risk factor (Appels & Mulder, 1988).

Separate analyses of each single item showed that 24 of the 57 items were predictive of future myocardial infarction when adjusted for age:

often tired
have trouble falling asleep
often wake up during the night
feel weak
feel as though I am not accomplishing much
have difficulties coping with everyday problems
believe to have come to a 'dead end'
feel listless
sexual interest lessened
feel hopeless
have difficulty in grasping a new problem
easily irritated
want to give up trying
feel fine (no)
body is like a battery that is losing its power
want to be dead at times
just don't have what it takes anymore
feel dejected
feel like crying
wake up exhausted
have difficulty in concentrating
have strange bodily sensations
shrink from regular work
have spells of shaking and trembling all over

The item analysis was completed by an analysis of the age-adjusted relative risk for each year of the follow-up. Most of these 24 items were found to have quite stable relative risks, averaging around 2.00. However, some items had remarkably decreasing risk ratios (Table I). These items reflect a loss of vitality and strong feelings of irritation or demoralisation. If DSM-III criteria were applied to the set of predictive items listed above, it should probably be concluded that the RCSS has provided some evidence that the onset of myocardial infarction is preceded by depressive feelings. This corresponds with the findings of other studies, which observed that especially patients dying of myocardial infarction passed through one or more periods of distress, exhaustion, or depression in their lives (Shekelle &

TABLE I

Age-adjusted relative risk of myocardial infarction associated with five mental complaints during four years of follow-up

Items	*Year of follow-up*			
	1	*2*	*3*	*4*
Not accomplishing much	4.53**	2.80**	1.89*	1.37
Easily irritated	5.88**	1.90*	1.53	1.40
Want to be dead	7.65**	4.32**	3.60**	2.90**
Strange bodily sensations	3.30**	1.04	.91	1.04
Shrink from work	3.01**	1.49	1.39	1.33

*$P<0.05$, **$P<0.01$.
Data from the RCSS (Appels & Mulder, 1988).

Ostfeld, 1965; Brozek *et al*, 1966; Paffenbarger *et al*, 1966; Lebowitz *et al*, 1967; Greene *et al*, 1972; Klatsky *et al*, 1976; Talbot, 1977; Lloyd & Cawley, 1983; Murphy *et al*, 1988).

A statement like "depression increases the risk of myocardial infarction" would be a parsimonious formulation for summarising the findings of many studies. Such a statement is not false, but it is rather crude. Important elements of the mental precursors of myocardial infarction might go unnoticed or could be misinterpreted if the most economical formulation is chosen.

The set of predictive items includes some known characteristics of depression, such as a sad, apathetic mood, loss of sleep and sexual desire, fatigue, and tiredness. Two other major elements of depression, a negative self-concept and feelings of guilt, do not belong to the precursors of myocardial infarction. None of the items about lowered self-esteem or feelings of guilt were found to be predictive in the RCSS. Self-reproach is rare among coronary victims. Typically, coronary patients blame others for the occurrence of negative events (Byrne, 1980).

The item with the strongest predictive power in the RCSS was 'Do you want to be dead at times?' When we formulated this item, we believed that it related to depression; in fact discussions with coronary patients who had endorsed that item showed that a positive answer was often chosen to express very strong feelings of annoyance or irritation. Increased irritability is a major component of the precursors of myocardial infarction. Next to symptoms of lack of energy (such as decreased participation in social activities like birthdays or celebrations), increased irritability is often mentioned by relatives of coronary victims when asked which behavioural changes they noticed in the months preceding the coronary event.

Because lowered self-esteem and feelings of guilt are not among the precursors of myocardial infarction, and in order to fit the description of this state into the description given by cardiologists, we have chosen the label 'vital exhaustion' for describing the construct measured by the MQ. Labels should communicate something, and this label fits well into the self-descriptions of coronary patients, who seldom use words like 'sad' or 'depressed' to describe their condition, but often prefer metaphors like 'my battery lost its power' or 'the well was drying up'.

The origins of vital exhaustion

Cardiological views

Many patients feel much better and lose their feelings of fatigue after bypass surgery or angioplasty. Therefore, many cardiologists are inclined to attribute the feeling of tiredness to the impaired oxygen supply to the myocardium or to side-effects of medication, especially diuretics and beta-blockers. Few, if any, cardiological studies have directly addressed the question of the origin of excessive tiredness before myocardial infarction.

Freeman & Nixon (1987) observed an association between some symptoms of exhaustion (especially 'waking up unrefreshed from sleep' and 'poor energy levels') and the number of symptomatic episodes of ischaemia during ambulatory monitoring. These authors note:

> "Even the shorter periods of ischaemia which do not cause infarction are still highly undesirable, because they can impair myocellular function and cause what Braunwald has termed the stunned myocardium. This depresses left ventricular functions and may be the main reason why many patients report a poor energy level when due inquiring is made."

This finding is important. However, the observed association can be explained in two ways: ischaemic periods cause poor energy levels or vice versa.

Sleep problems form part of the syndrome of vital exhaustion, and may be understood as a cause, an effect, or as a reinforcing element. Sleep problems and sleep apnoea have been reported as a risk factor for coronary heart disease by several authors (Siegrist, 1987). Sleep apnoea has a prevalence of 5–15% in middle-aged male populations. It facilitates the development of arterial hypertension and of left ventricular hypertrophy independent of volume load. Sleep apnoea increases the vulnerability of the myocardium to ischaemic attacks and, subsequently, to angina and acute myocardial infarction. It is therefore possibly a confounding factor in the association between vital exhaustion and future coronary heart disease. Given this possibility and the possibility that early unrecognised ischaemia may result in poor energy levels, there is no suggestion that vital exhaustion is a cause of coronary heart disease.

Psychodynamic views

One of the earliest psychiatric studies of the personality characteristics of myocardial infarction patients was conducted by Kemple (1945). His description is quoted at some length because it contains many elements that have been studied during the past two decades.

> "The subject is extremely ambitious, he generalizes compulsively, has good organizing ability. He is active and aggressive but productivity is inhibited by anxiety.

> Inner creativity is extremely limited, the inner life being dominated by infantile instinctive drives. Strong hostile aggressive impulses associated particularly with sex arouse feelings of guilt and of inner tension as he attempts to repress them. Emotional responses to the outer world are limited and when given are violent, uncontrolled and diffuse. He lacks subtle adaptive responses, and sensitivity to nuances in his environment. Moods and feelings are not stable, however. He is highly reactive, displaying infantile aggressiveness, oral dependence, guilt, depression and anxiety regarding his adjustment. . . . [He] strives compulsively to achieve goals incorporating power and prestige. Limitation of the introversive experiences' of creative thought and imaginative reflection increases the dependence upon achievements in the external world for satisfaction and security. . . . Although they try to keep their strong aggressive impulses under control, they manage to justify to themselves a good deal of outwardly expressed hostility."

Kemple's description influenced Friedman in his description of the aggressive, ambitious, and impatient 'type A'. Friedman *et al* (1987) speculated that persistent struggle resulted in a chronic state of hyperarousal, both physiological and behavioural, which manifested itself in the way in which individuals acted (e.g. rapid, abrupt speech), what they said (e.g. critical comments about others), and, presumably, in what they thought (e.g. perceiving others as challenging their control of situations). Furthermore, he reasoned that underlying such behaviours was a basic profound feeling of insecurity, a pervasive doubt about the level of their value in certain contexts, such as at home or at work. This insecurity seemed to be linked to a fear that the person was not genuinely respected and admired by others, and heightened arousal was associated with a constant striving to seek the symbols of recognition and reward as a way of allaying what appeared to be basic fears of inadequacy and insecurity. Hence, the type-A behaviour pattern served to diminish feelings of insecurity or self-doubt and was strongly reinforcing (Thoresen *et al*, 1985).

The type-A behaviour pattern has been found to double the risk of myocardial infarction, while attempts to change this behaviour pattern have resulted in a decreased risk of recurrent myocardial infarction (Rosenman *et al*, 1976; Friedman *et al*, 1987). The predictive power of the type-A behaviour pattern was confirmed in the Framingham study and in numerous case-control studies (Haynes & Feinleib, 1982; Matthews & Haynes, 1986). However, a prospective study, which used a structured interview to assess type-A behaviour, could not replicate the original findings (Shekelle *et al*, 1985). Furthermore, Ragland & Brand (1988) observed that type-A behaviour is inversely related to survival after myocardial infarction.

It is hard to integrate the old findings with the recent ones. Any attempt to do so would be beyond the scope of this paper. Suffice it to say that in recent years several scientists have directed their attention to one of the behavioural components leading to susceptibility to coronary problems mentioned by Kemple, namely hostility. This factor was found to be the strongest predictive component of type-A behaviour in the Western Collaboration Group Study and was also found to be associated with future coronary heart disease in other studies (Matthews *et al*, 1977; Shekelle *et al*, 1983).

Limitation of creative thought and imaginative reflection was found to be predictive of future coronary heart disease in a study by Bonami & Rimé (1972). They observed that future coronary patients' responses to Van Lennep's four-picture tests were less rich in fantasy and imagination, and reflected more ambivalent conflicts in relations with other males than the responses of a control group who remained free of coronary heart disease during a ten-year follow-up. High reactivity is now mainly studied by psychophysiologists who assume that 'hyperactivity' forms the link between type-A behaviour and coronary heart disease (Matthews & Haynes, 1986). Other elements of Kemple's description, such as the lack of subtle adaptive responses, deserve detailed investigation, but this has yet to be carried out.

Arlow (1945) interpreted coronary patients' compulsive striving as a result of an unsuccessful identification with their father. According to Arlow the usual story of the patient with coronary artery disease is that of a stubborn, self-willed child who enters a competitive relationship with a much-feared and envied parent at an early age. The conflict is then repressed and identification with the parent takes place. Through working hard, self-discipline, and compulsive devotion to the completion of tasks which he assigns himself, the patient keeps driving himself to success. However, he is constantly drawn into new situations in which he repeatedly recreates the original competitive situation, a situation which he attempts to master by identifying with his superiors. The adaptive mechanism of compulsive striving for achievement and mastery starts to fail in the period before the onset of myocardial infarction.

The first quantitative psychiatric study of the short-term precursors of myocardial infarction was published by Hahn (1971), who observed that coronary patients were significantly more compulsive, involuntary counters of trivial things, like houses or trees, than controls were. He interpreted this compulsive behaviour as a defence mechanism directed against a depressive ambivalence conflict. Upon decompensation of this defence through conflicts or stress, the underlying anxieties and depressive way of coping with conflicts manifested themselves. By means of a questionnaire, Hahn looked into the ways in which the decompensation was experienced by coronary patients. The vast majority reported to have experienced increasing irritability, strong mood changes, increased sweating, and the general feeling of not being well (Hahn, 1971).

Psychological views

On the basis of the type-A theory, Glass (1972) developed a psychological model in which type-A individuals, who suffered from coronary heart disease, passed a state of frustration and exhaustion preceding myocardial infarction:

> "If the type A individual concludes that little can be done to rectify the situation, we may expect him to experience more intense feelings of helplessness than are experienced by a type B in similar circumstances. This prediction is derived from the assumption that type A's probably try harder than type B's to avoid losing their jobs when that possibility first becomes apparent. Exerting greater initial efforts at control, type A's experience greater helplessness when they become convinced that nothing can be done about being fired.
>
> The precise role of initial hyperresponsiveness and subsequent hyporesponsiveness (helplessness) in the development of cardiovascular pathology remains unclear . . . we should first determine whether the interaction of Pattern A and helplessness is indeed prodromal to clinical coronary heart disease."

Some evidence supporting the model of Glass is provided in a study by Falger (1989), who compared 133 males, admitted because of a first myocardial infarction, 192 hospital controls, and 133 neighbourhood controls, matched for age. This study included an assessment of vital exhaustion, as experienced in the months preceding the coronary events, by the MQ, an assessment of type-A behaviour by means of a structured interview, and an interview about major live events. To test the hypothesis that exhausted type-A persons are at increased risk compared with vital type-A subjects, Falger divided cases and controls into vital or exhausted type As and type Bs. Seventy-five coronary cases (56%) were classified as exhausted type-A persons, compared with 32% of the hospital controls and 24% of the neighbourhood controls. These differences are highly significant. Compared with vital type-B persons, exhausted type-A persons had a fivefold greater estimated risk in the hospital series and an elevenfold greater estimated risk in the neighbourhood series. When exhausted type-A people were compared with vital type-A persons, the estimated relative risk was 2.67 in the hospital series and 5.52 in the neighbourhood series.

Feelings of vital exhaustion were found to be positively associated with a number of life events or more chronic aversive conditions, such as 'serious conflicts at work with supervisor(s)', 'serious conflicts at work with subordinates', and 'prolonged overtime work'; 'serious educational problems with children', 'serious marital conflicts', and 'prolonged financial problems'. The strongest association was between vital exhaustion and 'prolonged overtime work', which discriminated significantly between cases and controls, but lost its discriminating power when vital exhaustion was controlled for. Thus, overwork is a risk factor for coronary heart disease when it leads to exhaustion. This finding also indicates that feelings of exhaustion are not only caused by uncontrollable events or other forms of mental stress, but may also be caused by more physical factors like double jobs or too many working hours, which deprive people of sufficient time spent relaxing.

Some unresolved questions

Falger's (1989) data suggest that vital exhaustion results from distressing experiences or work overload. Such an interpretation of the origins of vital exhaustion corresponds with the attribution of the causes of these feelings by coronary patients. However, it could also be argued that negative experiences are especially distressing to those who already felt weak and tired. Therefore the direction of the association between life events and vital exhaustion still is unclear.

Little is known about whether vital exhaustion reflects a more or less permanent personality feature or a more transient state of stress. The RCSS data indicate that vital exhaustion is not a stable personality characteristic. The test–retest stability of the MQ was found to be 0.30 when retesting was done after four years among a random subsample of 92 participants. Another finding of the RCSS indicated that memories of earlier periods of mental and physical exhaustion are reactivated before myocardial infarction. During the clinical interviews designed for developing the MQ, many coronary patients reported that they had been 'overwrought' once or several times in their lives. They reported that their mental state before admission was almost similar to that earlier condition. For that reason the item 'Have you ever been overwrought?' was added to the MQ. A positive answer was found to be associated with an increased risk of myocardial infarction, the age-adjusted relative risk being 1.81 (95% CI 1.10–2.99). A positive reply was also strongly correlated with the predictive items, suggesting that an earlier period of breakdown in adaptation was associated with the current condition.

Hardly anything is known about mechanisms which could explain the association between vital exhaustion and coronary heart disease. Van Doornen (1988) observed that exhausted subjects reacted to mental stress with increased levels of cholesterol and noradrenaline. This reaction might be especially harmful in people with a somatic predisposition to myocardial infarction. We do not believe that vital exhaustion is a necessary or a sufficient cause of coronary heart disease, but we do believe that this state is like a powerful wind which may stir up an already existing fire.

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Comment

Confidence – and clinical importance – in research findings

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Testing the null hypothesis has been the cornerstone of much statistical theory and practice over the last few decades. During this time it has encroached on most disciplines and become widespread in use within them. There are now few research papers in the scientific literature where the outcome of one or more hypothesis tests is not reported. With this 'progress' there has undoubtedly been an improvement in the way in which data have been collected, analysed, and interpreted, and a move towards more objective evaluation of study results.

However, all is not rosy. The understanding of what is actually involved in testing the null hypothesis and the interpretation of the outcome (in terms of the *P* value) is far too hazy. This is not really surprising since tests of the null hypothesis begin by turning an investigator's idea (for example, that some new treatment will perform better than that currently being prescribed) upside down and substitute the notion of no effect or no difference. The test then evaluates the probability of the observed study result, or a more extreme result, occurring if this null hypothesis were in fact true. Awareness that this is what the *P* value measures is all too rare. Also, proper understanding of a study result is obscured by transforming it from the study scale of measurement onto a remote scale constrained from zero to unity. Nonetheless, obtaining a low *P* value, particularly <0.05, is widely interpreted as implying merit, in some abstract sense, and leads to the findings being generally accepted as important and deemed publishable. In contrast, this status is often denied study results which have not achieved this magic, but arbitrary, characteristic.

A move away from the dominance of hypothesis testing has emerged over the last few years and, after a suitable gestation period, is gaining wider acceptance. The change has been towards the use of estimates of, for example, the differences in outcome between treatment groups together with confidence intervals. The basic premise is simple and attractive. It encourages the evaluation and reporting of study results on the original scale of measurement, while simultaneously incorporating some information on their precision or imprecision. In this way the author and reader are led to an interpretation of the study results which directly involves an assessment of the numerical size of the comparison of interest. In addition, there is explicit recognition that a single study can give only an estimate of any true (treatment) effect as a consequence of sampling error. This approach focuses thinking much more along lines of clinical importance rather than statistical significance. The latter is often used to provide a lazy dichotomy into positive or negative results of studies, when, of course, there is really no such division, certainly at least not where $P=0.04$ puts a study into one category and $P=0.06$ puts it into the other.

The particular technique recommended to enable a suitable presentation, in terms of demonstrating (im)precision, of a study's results is the 'confidence interval'. Its use has now been advocated in a number of medical journals (Rothman, 1978; Gardner & Altman, 1986; Berry, 1986; *Lancet*, 1987; Braitman, 1988; Evans *et al*, 1988; International Committee of Medical Journal Editors, 1988). The rationale for confidence intervals as well as methods for their calculation for many common statistical procedures, such as means, medians, proportions, and regression lines (and in each case for their differences), have been described, with examples, in "Statistics with Confidence" (Gardner & Altman, 1989). The basic building blocks are well known (for example, the mean difference in outcome between treatments together with its standard error) from which a confidence interval is easily calculated. The precise method, however, varies. An easily used computer program (CIA) dedicated to calculating confidence intervals is available through the *British Medical Journal*.

The thinking behind this revised philosophy comes mainly, as said, from the present overemphasis on statistical tests of the null hypothesis, together with the desire to take data analysts back to looking at their results in a more immediately appealing manner. It would be a return to a situation similar to that before statisticians became involved – but now including both the constraints and positive aspects of examining imprecision. The essence of the confidence interval is to provide a range of uncertainty, which will be wider for smaller samples, emphasising unreliability. In this sense it is especially useful for so-called negative studies, to indicate the range of possible true values. Confidence intervals thus give a measure of the precision of study sample results for making inferences about the population. They allow for statistical sampling error (deviation in the sample from truly representing the population) but cannot allow for non-sampling errors due to inappropriate research methods.

A look through the *Journal* reveals a high profile for statistical hypothesis testing rather than estimation. As examples of current practice and to illustrate the advantages of confidence intervals, we will discuss two papers from recent issues of the *Journal*. First, a paper which examined pre-morbid intelligence in some organic conditions compared 70 patients with controls matched for age, sex, and duration of education (Crawford *et al*, 1988). Two psychometric tests, based on reading and vocabulary, were administered and the results converted to full-scale IQ scores. Comparisons were then made between patients and controls – for example, mean IQ scores were compared using the paired *t*-test and found to be significantly different ($P < 0.001$). The paper did not directly report the actual mean differences between the groups in IQ scores, and assessment was based mainly on the statistical significance or otherwise of the differences, with little comment being made about their clinical importance.

How could confidence intervals help? For example, the size of the mean difference in vocabulary-estimated IQ between controls and patients in this study was 7.2 points – but how precise is this figure? A confidence interval can tell us. Although no estimates of variability (for example, standard deviations) are given, it is possible from the published paper to calculate a confidence interval for the mean difference in vocabulary-estimated IQ indirectly through the result of the reported *t*-test. This gives a 95% confidence interval of from 4.9 to 9.5 IQ points, indicating the degree of precision in the estimated mean difference of 7.2 IQ points.

Second, Ryan *et al* (1989) examined the psychological state before and after hysterectomy of 60 women who underwent the operation for benign conditions. They report that 55% (33 of 60) pre-operatively and 32% (19 of 60) post-operatively of the women could be described as 'psychiatric cases' (mainly depression), and that this decrease was statistically significant ($P < 0.01$). From information given in Fig. 1 of the published paper it is possible to calculate a confidence interval for this 23% decrease, taking into account the before/after feature of the study design. The resultant 95% confidence interval for the true decrease is found to be from 8% to 31% (using the exact method described in Chapter 4 of Gardner & Altman (1989)). These figures imply that there is a range of uncertainty about the degree of reduction in depression to be expected following hysterectomy on the basis of this one study – it could be substantially smaller (near the lower values of the confidence interval – 10% or less) or greater (30% or more) than the actual study result of 23%. Put simply, the meaning of this confidence interval is that there is a 95% chance that the indicated range includes the 'population' decrease in level of depression – that is, the value that would be obtained by studying the whole population of women having hysterectomies about whom inferences from the study are aimed. These relatively large potential differences in outcome are hidden by the bland statement of the actual observed reduction and its statistical significance.

It seems to us appropriate that the *Journal* should join others by reducing the attention given to the level of statistical significance of study results and increasing the use of estimation and confidence intervals. The potential benefits of the latter were previously indicated in section 3.4 and elsewhere of the statistical guidelines (Altman *et al*, 1983) that are strongly advised for authors by the *Journal* in its "Notice to Contributors" (these have been slightly revised by Gardner & Altman (1989)). The recommended approach is to present an estimate of the comparison of interest, for example, the difference between the proportions of patients responding to treatment, together with a confidence interval to indicate the imprecision of this estimate based on the results of a single study. The *P* value, on its own, tells us nothing about the magnitude, or even the direction, of any difference between treatments. We do not suggest that only confidence intervals be given, nor that confidence intervals should be given for every comparison. We do suggest, however, that confidence intervals accompany the main results of a study.

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Psychiatric Emergencies, Scotland and the World Cup Finals

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Reductions in emergency psychiatric presentations to hospital occurred during and after the finals of the World Cup football competitions, an effect evident in women as well as men, and more marked among the mentally ill than those who were not. Increases in the numbers of schizophrenic and neurotic men presenting before, and alcoholic men during, the competitions can be attributed to the football. The generalised reductions during and after could reflect an indirect effect upon help-seeking, although actual relapse rates of mental illnesses might be affected. These changes could arise from enhancement of national identity and cohesion.

The finals of the four World Cup football competitions since 1974 in which Scotland participated have consistently coincided with a fall in the frequency of parasuicide in both sexes, which has been sustained for at least eight weeks after the last game (Masterton & Strachan, 1987). We now report what happens to the frequency and nature of another form of psychosocial crisis, emergency psychiatric presentations to hospital, before, during, and after the tournaments.

Method

Since July 1977 systematic information upon all emergency and out-of-hours presentations to the Royal Edinburgh Hospital (with a catchment population of 470 000) which serves the entire city, has been collected for the Lothian Psychiatric Case Register. This source provided data upon all cases seen around the 1978 (26 days), 1982 (30 days), and 1986 (30 days) finals of the World Cup. Intervening years acted as controls, using the corresponding dates in 1979/80 for 1978, 1981/83 for 1982, and 1984/85 for 1986; the 1982 tournament began two weeks later than those in 1978 and 1986, which started in the first week in June.

The method adopted was to aggregate the three World Cup years and test for differences from the combined six control years, based upon the assumption that the numbers of cases in each time interval should represent the same proportion of the annual totals. The percentage difference between World Cup and other years was evaluated statistically by calculation of the 95% confidence interval.

This technique was applied to the number of emergency presentations in total and by sex during the competitions and in the two four-week periods before and three four-week periods afterwards. Then changes within diagnostic categories were examined, confining this analysis to the commonest primary diagnoses and four weeks either side of the tournaments.

Table I

The number of psychiatric emergencies and percentage difference in proportion of the annual total: World Cup v. other years

	−8 to −4 weeks	*−4 to 0 weeks*	*World Cup*	*0 to +4 weeks*	*+4 to +8 weeks*	*+8 to +12 weeks*
Males						
n: World Cup (other) years	272 (535)	231 (446)	238 (524)	210 (543)	225 (552)	252 (457)
% difference	+5%	+7%	−6%	−20%	−16%	+14%
95% CI	−7 to +17%	−6 to +21%	−18 to +6%	−32 to −8%	−27 to −4%	−1 to +27%
Females						
n: World Cup (other) years	188 (401)	197 (409)	180 (422)	191 (417)	191 (504)	179 (382)
% difference	+2%	+5%	−7%	−1%	−18%	+2%
95% CI	−12 to +16%	−10 to +19%	−21 to +6%	−14 to +13%	−30 to −6%	−13 to +16%
Total						
n: World Cup (other) years	460 (936)	428 (855)	418 (946)	401 (960)	416 (1056)	431 (839)
% difference	+4%	+6%	−7%	−12%	−17%	+9%
95% CI	−6 to +13%	−4 to +16%	−16 to +3%	−21 to −3%	−25 to −8%	−1 to +19%

Results

Frequency

Before the tournaments there were more presentations than expected, but this fell during the finals and substantially further in the subsequent eight weeks. The effect was more marked in males, who had 14% fewer presentations in the eight weeks after the tournaments compared with before, against a 12% increase in control years. The fall in females was principally due to changes in control years (+14%), with only a 1% drop between the same periods in World Cup years.

The reductions occurred consistently after each competition but not during (*during*: 1978 = −19%, 1982 = −2%, 1986 = +3%; *0 to +4 weeks*: 1978 = −7%, 1982 = −9%, 1986 = −17%; *+4 to +8 weeks*: 1978 = −28%, 1982 = −9%, 1986 = −14%).

That these effects were not due to other differences between the years nor subsequently compensated for is indicated in Table II. World Cup years differ significantly for annual totals but not when the period during and after the tournament is excluded.

Diagnosis

Of 4008 emergency presentations during the 12 weeks under consideration in the years 1978–86, 3288 (82%) were accounted for by alcoholism (736 males, 183 females), schizophrenia (382, 225), affective psychosis (187, 329), neurosis (125, 242), and no mental illness (378, 501). The last grouping comprised personality disorder, adjustment reaction, and no psychiatric disorder, and reflects a suggestion for a hybrid category of uncertain illness status discussed in connection with ICD–10 (Cooper, 1988).

The annual totals for all specified diagnostic categories differed by 2% or less from expected in World Cup years, indicating a general stability in the frequency of presentation and diagnostic system.

This was not the case for the remainder of the diagnoses, where the annual total in World Cup years was significantly less than expected. Part of the explanation may lie in changes in the classification introduced in 1983, but probably more important were developments in the management of some categories of emergency, notably drug dependence and organic states.

The main findings were excess presentations of schizophrenia before the tournaments, and reductions in all illness categories during and afterwards (with the exception of alcoholism during), which contrasts with lack of change in the non-illness category. The statistically significant differences by sex were increases before the tournament in male schizophrenics (+65%) and neurotics (+133%), and reductions during of female affective psychosis (−35%), and after of male neurotics (−56%).

The commonest presentation, males with an alcohol problem, was perhaps the most interesting, with a 19% excess of presentations during the competitions, not quite statistically significant (95% CI −4 to +41%) but sufficiently influential against the trend to explain why the overall decrease was less during the World Cup than in the subsequent eight weeks. Indeed, with male alcoholics discounted the reduction during the World Cup becomes statistically significant not only for males (−18%, −31 to −4%) but also overall (−12%, −22 to −2%).

Discussion

We are confident that our main finding, a reduction in the numbers of emergency psychiatric presentations during and after World Cup final competitions, is a valid result. We feel more circumspect about the findings within diagnostic groups, our method being

TABLE II

The total number of psychiatric emergencies in World Cup (other) years with percentage difference for World Cup years v. all years, 1978–86

	Annual totals	*Annual totals excluding contribution from World Cup finals and subsequent eight weeks*
Males		
n: World Cup (other) years	2766 (5724)	2093 (4105)
% difference from expected	−2.3%	+1.3%
95% CI	−5.3 to +0.8%	−2.3 to +4.9%
Females		
n: World Cup (other) years	2235 (4853)	1673 (3510)
% difference from expected	−5.4%	−3.2%
95% CI	−8.7% to −2.1%	−7.0 to +0.7%
Total		
n: World Cup (other) years	5001 (10577)	3766 (7615)
% difference from expected	−3.7%	−0.7%
95% CI	−5.9 to −1.5%	−3.4 to +1.9%

TABLE III

The number of psychiatric emergencies and percentage difference in proportion of the annual total: World Cup v. other years and World Cup v. all years for the annual total

	Schizophrenia	*Affective psychosis*	*Neurosis*	*Alcoholism*	*No mental illness*	*Other diagnoses*
Before (−4 to 0 weeks)						
n: World Cup (other) years	76 (104)	57 (115)	45 (78)	89 (196)	96 (198)	65 (164)
% difference	+51%	+3%	+17%	−7%	−5%	−1%
95% CI	+23 to +79%	−23 to +29%	−15 to +48%	−26 to +12%	−24 to +14%	−25 to +23%
During World Cup						
n: World Cup (other) years	60 (140)	48 (120)	31 (88)	114 (200)	95 (185)	70 (213)
% difference	−11%	−17%	−29%	+17%	+1%	−18%
95% CI	−34 to +12%	−41 to +7%	−56 to −2%	0 to +33%	−19 to +21%	−36 to 0%
After (0 to +4 weeks)						
n: World Cup (other) years	69 (158)	55 (121)	35 (90)	92 (228)	101 (204)	49 (159)
% difference	−10%	−6%	−21%	−17%	−3%	−23%
95% CI	−31 to +12%	−30 to +19%	−49 to +6%	−35 to 0%	−21 to +16%	−46 to 0%
Annual totals						
n: World Cup (other) years	760 (1573)	675 (1400)	461 (931)	1137 (2329)	1080 (2123)	888 (2221)
% difference from expected	−2%	−2%	−1%	− 2%	+1%	−14%
95% CI	−8 to +4%	−9 to +4%	−8 to +7%	−7 to +4%	−4 to +7%	−20 to −9%

less stringent for two reasons. Firstly, the number of patients admitted over any four weeks was small in some categories, resulting in imprecision expressed as a wide confidence interval. Secondly, the inter-rater reliability for the diagnostic system is unknown, although it is likely to be satisfactory given its basis of simplification and abbreviation from the *International Classification of Diseases*. Additionally, the area of poorest reliability (with DSM-III at least) is in differentiating personality disorder, adjustment reaction, and no psychiatric illness (Williams & Spitzer, 1980), and of course this was avoided in our analysis by combining these categories.

It seems logical that a period of anticipation and increasing excitement, as in the weeks leading up to an important event, will represent another stress upon the mentally vulnerable, and the nature of this event, football matches, probably explains the increase in schizophrenic and neurotic presentations being largely confined to men. Similarly, the increase in alcohol-associated male presentations during the tournaments surely reflects a direct, immediate consequence of excessive alcohol consumption at such times, and the association of Scotsmen and alcohol with football matches ought to come as no surprise.

The firm finding of reduced emergency psychiatric presentations during and after the tournaments, which accords with the changes reported for parasuicide (Masterton & Strachan, 1987), has a less obvious explanation, and the fact that both sexes are affected for several months after the final whistle suggests that this is not directly attributable to football. There are few outlets which permit a wide and acceptable expression of Scottish nationhood – sport is perhaps the most powerful, and football is the national game. The World Cup finals enhance a sense of national identity as well as representing a focus of purpose and excitement, and it was this that prompted our investigations. We would speculate that such a common interest and endeavour, fused with a surge of nationalism, might enhance social cohesion in the manner proposed by Durkheim (1952) to explain the decreased suicide rates that accompany times of war.

Why this effect should be more pronounced in parasuicide and those who are mentally ill, rather than those who are not, is intriguing. If this is indeed the case it suggests complex changes in help-seeking among people in crisis. Perhaps people are less liable to be introspective or to despair, more likely to tolerate and cope, and to deal with any crisis more directly. While strategies adopted in crisis probably change, it is also possible that the reductions observed across a range of mental disorders could be due to a direct action upon the rate of relapse, given that social factors are known to influence such recurrences (Leff & Wing, 1971).

In conclusion, our findings indicate that national participation in the World Cup final football competitions exerts a broad and stabilising effect upon human behaviour which extends into the field of primarily biologically determined mental illnesses. Whether physical health is also affected has not been established – the only study retrieved in a comprehensive literature search (Rost *et al*, 1975) reported increased risks of cardiac arrhythmias and high blood pressure in West German men watching the 1974 tournament – but then they were winning!

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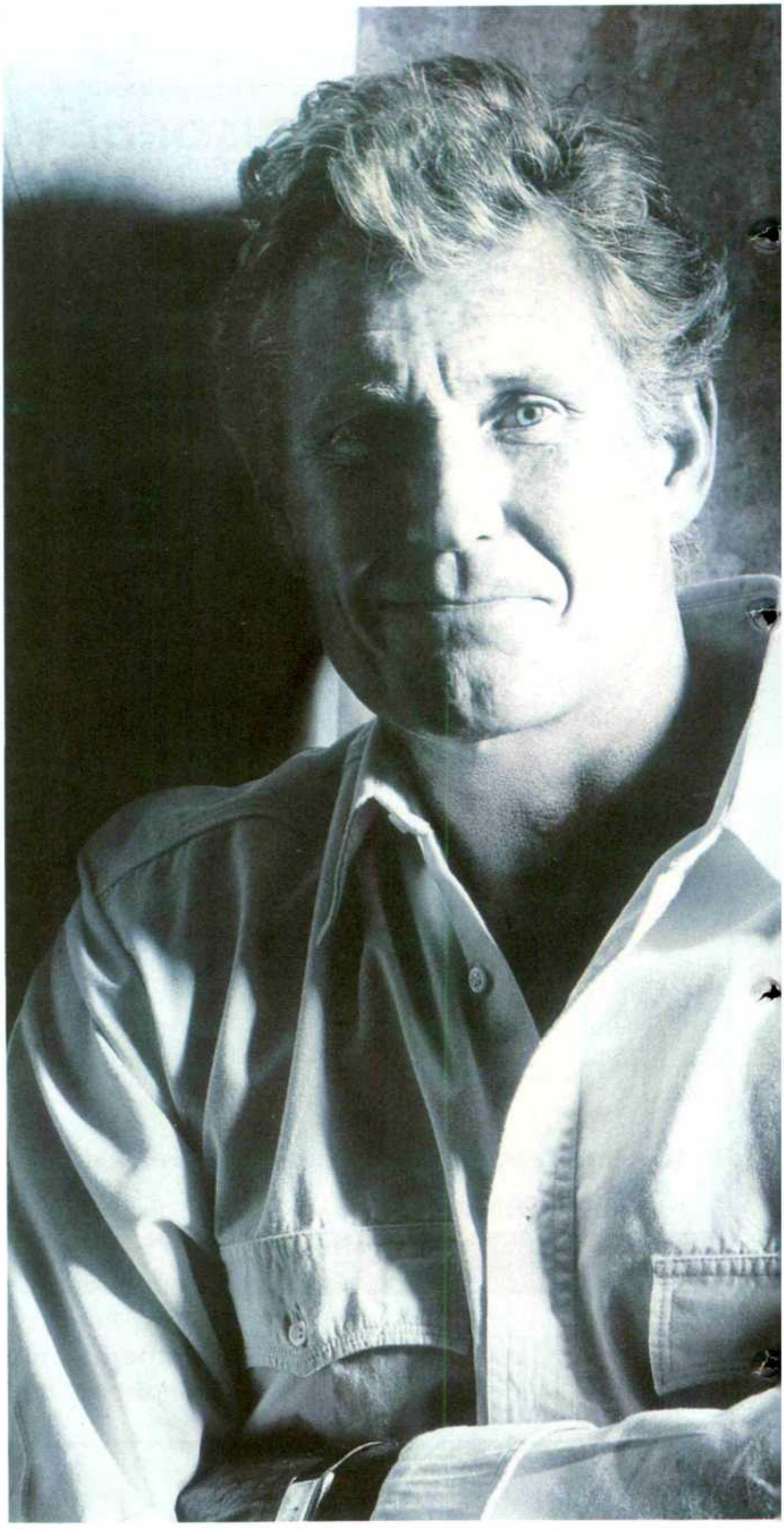

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"Insane ear seems to have been commonest in general paralytics, and its disappearance, from the literature at least, coincided with the decline of general paralysis. Yet general paralysis continued to be a common disorder for decades after insane ear had apparently gone." Edward Hare, Chapter 6, Old familiar faces: some aspects of the asylum era in Britain

"This English disease, affecting, he insisted, as many as one-third of all people of quality in England, was the result of high living, prosperity, and progress unique on such a wide scale in early 18th-century England. Cheyne flattered his patients: only those of quick and lively dispositions and good breeding suffered from the nerves." William F. Bynum, Chapter 8, The nervous patient in 18th- and 19th-century Britain. the psychiatric origins of British neurology

"Abnormal childhood sexuality was an equally strong clue to the presence of 'chronic sepsis and toxemia ... [and] in a number of cases the colon has been resected with improvement in the individual'." Andrew Scull, Chapter 10, Desperate remedies: a Gothic tale of madness and modern medicine

"Claude Bernard ... opposed ... the view that averages or indeed statistics could ever lead to exact science." German Berrios, Chapter 12, Memory and the cognitive paradigm of dementia during the 19th century: a conceptual history

British Journal of Psychiatry (1990), **156**, 479–482

Psychological Consequences of the Enniskillen Bombing

P. S. CURRAN, P. BELL, A. MURRAY, G. LOUGHREY, R. RODDY and L. G. ROCKE

Eleven people were killed and 60 injured in the Enniskillen bombing of November 1987. Survivors were psychologically appraised six months and one year later. At six months 50% had developed post-traumatic stress disorder (PTSD). This group comprised more females than males. However, all victims had high scores on the GHQ. We found no correlation between psychological injury (as measured by the GHQ) and physical injury (as measured by the ISS), calling into question previous assertions.

On 8 November 1987 a crowd gathered at the Cenotaph in Enniskillen to celebrate Remembrance Sunday. A bomb exploded without warning in an adjacent building, crushing bystanders. Eleven people were killed and some 60 injured. The rescue scenes were captured on amateur video and transmitted across the world.

Six months later 33 survivors were referred to our department for medico-legal assessment. Each had been in the square at the time of the bombing and none was more than 30 metres away from the explosive device. Physical injuries ranged from minor cuts and bruises to fractured pelvis, fractured skull, or ruptured spleen. Victims of violence are entitled to seek compensation under the Criminal Injuries Order 1977 (Northern Ireland). We have previously commented (Kee *et al*, 1987; Curran, 1988; Loughrey & Curran, 1977) upon the methodological difficulties in identifying the actual victims of violence, and have documented our clinical findings and experience in assessing psychological injury following violent incidents of many different types in Northern Ireland, ranging from assassination attempts to common assault (Kee *et al*, 1987; Loughrey *et al*, 1988; Bell *et al*, 1988). The Enniskillen atrocity afforded a unique opportunity in that, while previously we had reported retrospectively on victims of uniquely different experiences, in this case all victims had witnessed the same experience and were all examined exactly six months and one year after the incident.

Method

A psychiatric history and examination were conducted by a consultant psychiatrist, and relatives' accounts considered. Each victim completed the General Health Questionnaire (GHQ; Goldberg, 1978), using the usual 0–1 scale for scoring, while two other psychiatrists independently applied a schedule to determine which victims were suffering from post-traumatic stress disorder (PTSD). This is a specific type of psychological reaction to stress as defined in DSM–III–R (American Psychiatric Association, 1987) which provides a different dimension and another way of looking at psychological injuries. It has been applied after a variety of disasters around the world, as we have discussed in our previous papers (Bell *et al*, 1988; Loughrey *et al*, 1988).

In each case an ICD–9 diagnosis (World Health Organization, 1978) was also reached by the examining psychiatrist. The value of ICD–9 in categorising post-traumatic reactions is limited. The code 308 describes acute reactions to stress – transient disorders of any severity and nature following exceptional physical or mental stress, such as a natural catastrophe or battle. We use it here to quantify reactions of less than two to three weeks' duration. The code 309 describes adjustment reactions – again, transient disorders, quite circumscribed, reversible and situation-specific, lasting up to four months. It has been our previous experience (Kee *et al*, 1987) that approximately one victim in three has such a reaction to acts of terrorist violence in terms of severity and duration of symptoms. If a victim showed little evidence of significant repair of mental symptoms by four months then they are described as suffering neuroses of whatever clinical subtype (code 300).

We sought to examine the relationship between physical and psychological injury. An experienced consultant in accident and emergency medicine independently and blindly surveyed the victims' contemporary hospital records and rated each victim's physical injuries using the Abbreviated Injuries Scale (AIS; *Journal of the American Medical Association*, 1971), and the Injury Severity Score (ISS; Baker, 1974). The ISS is the sum of the square values of the patient's three highest single AIS ratings. It is an accepted and well validated index of the severity of physical injury, useful in the epidemiological study of traumatic injuries (Baker & O'Neill, 1976; American Association for Automotive Medicine, 1985; Greenspan *et al*, 1985). In this scoring system an uncomplicated concussion, for example, has an AIS score of 2 and and ISS score of 4; an uncomplicated fracture of the ankle has an AIS score of 3 and an ISS score of 9; minor skin injuries are scored 1 on both scales. Thus, a person with three such injuries has a total ISS score of 14. An ISS score over 10 usually defines a patient with multiple or severe injuries (Malt, 1988). These results were analysed by χ^2 and Mann–Whitney U, Student's t-test and Spearman's ϱ.

Results

Of the 33 victims referred, seven children under the age of 14 were excluded from this study. Of the remaining 26, 13 were female and 13 male (mean age 30.15 years, s.d. = 13.89). ICD–9 diagnoses (Table I) ranged from acute reactions to stress through adjustment reactions to anxiety and depressive neuroses.

At six months 13 patients satisfied the criteria for PTSD (9 women, 4 men, $P<0.01$). Those with PTSD tended to be older (mean age 33.2 years compared with the whole group's mean age of 30.15 years), but this did not reach statistical significance. The PTSD patients tended to have higher GHQ scores (mean 20.37, s.d. = 6.93, v. mean 18.38, s.d. 6.52, for whole group), but this did not reach statistical significance. There was no significant correlation between GHQ score and age. The mean ISS score for those patients with PTSD was 1.2, significantly lower than 6.6 for the non-PTSD group ($P<0.01$). Interestingly, of the five seriously injured victims (i.e. ISS scores > 10), none had developed PTSD ($P<0.01$) at six months.

There was no significant correlation between physical and psychological injury (ISS and GHQ scores respectively) ($\varrho=0.136$).

At the second assessment, exactly 12 months after the incident, we found that of the original group who had active PTSD, only one had recovered. There were two patients with delayed PTSD, developing between six months and one year, both of whom had suffered severe physical injuries necessitating earlier hospital admission. Two patients who appeared to have recovered from PTSD at six months had symptoms again at the second assessment.

TABLE I

Details of the 26 patients referred for assessment

Group	*Age: years*	*Sex*	*PTSD* *Before 6 months*	*At 6 months*	*At 12 months*	*GHQ* *At 6 months*	*At 12 months*	*ISS*	*ICD–9 code*
Physically injured									
1	16	M	No	No	Yes	23	19	19	309.9
2	14	F	No	No	No	21	20	10	308.1
3	15	M	No	No	No	15	1	14	300.0
4	27	M	No	No	No	19	21	14	309.2
5	62	M	No	No	Yes	20	0	19	300.4
PTSD remission before 6 months									
1	36	F	Yes	No	Yes	25	20	1	309.2
2	15	F	Yes	No	Yes	20	23	2	309.2
3	17	M	Yes	No	No	23	0	0	309.2
4	34	F	Yes	No	No	14	16	1	309.2
5	29	M	Yes	No	No	1	6	1	308.4
Active PTSD at 6 months									
1	28	F	Yes	Yes	Yes	19	20	2	309.1
2	26	M	Yes	Yes	Yes	27	28	0	309.1
3	28	F	Yes	Yes	Yes	16	19	1	309.1
4	50	F	Yes	Yes	No	13	8	0	300.2
5	53	F	Yes	Yes	Yes	24	24	1	300.4
6	31	M	Yes	Yes	Yes	22	17	5	309.1
7	14	F	Yes	Yes	Yes	28	23	1	309.4
8[1]	34	F	Yes	Yes	–	14	–	1	300.4
Non-PTSD group									
1	47	F	No	No	No	26	1	0	309.2
2	17	F	No	No	No	16	21	1	309.0
3	25	M	No	No	No	6	0	0	309.2
4	38	M	No	No	No	8	16	1	309.2
5	57	M	No	No	No	23	27	5	309.9
6	25	M	No	No	No	17	14	1	309.2
7	23	F	No	No	No	16	18	1	309.1
8	23	M	No	No	No	22	14	1	309.0

1. Not followed up to 12 months

Apart from these findings the assessment of PTSD was remarkably consistent between six months and one year.

Discussion

The difficulties of studying a litigant population have been described elsewhere (Kee *et al*, 1987; Bell *et al*, 1988). There is always the difficulty that compensation issues will affect the findings, but because sampling after violent incidents and disasters has proved so difficult, the study of litigants is probably the most frequent sampling method to be reported. It is impossible to trace everyone who witnessed an incident of this nature and all sampling methods are subject to some form of bias. It would be desirable to compare our litigant population with a population who had witnessed the bombing but not sought compensation; however, this distinction in itself would lead to a sampling bias and further methodological difficulties. It is estimated that approximately 500 people were within 30 metres of the explosion.

The main risk is that symptoms may be over-reported for financial gain. However, this population was unaware of the strict diagnostic requirements for PTSD and could not have feigned the specific symptom sets. Clinically, we were impressed by the severity of the psychological suffering and the victims' tendencies to play it down. This clinical impression was supported by the very high GHQ scores for the group as a whole (mean 18.38), when a score of four or five usually represents a 'case' (Goldberg, 1978).

The only normative data for mental health in the general population in Northern Ireland comes from a study by Cairns & Wilson (1984). They surveyed two town populations, one with a high and one with a low incidence of civil disorder, using the GHQ. Although these authors marked the GHQ in a slightly different way, when we compared the GHQ scores found in our sample following the Enniskillen bombing with the community survey performed by Cairns & Wilson, we found that our sample had a mean GHQ score in excess of twice that which would be expected in a community survey in Northern Ireland.

In our earlier study looking at 643 victims of various types of violence in Northern Ireland (Bell *et al*, 1988), the PTSD group was more severely affected in terms of treatment uptake, duration of syndrome, and time off work. There was also a tendency for the PTSD group to be older and to contain more women. The results of the present study are in keeping with those findings (that higher scores on the GHQ and age differences did not reach statistical significance is possibly because of the smaller numbers). In our previous study (Bell *et al*, 1988) we identified 23% of the victims of different types of terrorist violence as suffering from PTSD. This is less than the 50% found in the present study, and may reflect the earlier assessment in the present study and the fact that this was a particularly horrific and stressful incident.

Miller (1961) suggested that psychological injury was inversely proportionate to physical injury. However, in this study ISS scores did not significantly correlate with GHQ scores. In fact, the tendency was towards a positive rather than negative correlation. We recognise that there was a bias towards high psychological disturbance, because of the selection of the sample, so that a correlation across the whole range of GHQ scores would be unlikely to emerge. When we examined the sample at six months we found that no seriously injured victim had the specific syndrome of PTSD. None of them were suffering at that time from decreased attention, nor were they in such a state of pain or other physical discomfort that they were inaccessible for inquiry concerning PTSD. Kennedy & Johnston (1975) have suggested that psychological symptoms can be delayed, particularly in the presence of physical injury. We would hypothesise that the intense attention and the cossetted hospital environment may protect the physically injured from PTSD and psychological disturbance in the initial stages, only to see it emerge when they are discharged from hospital. Our findings tend to support this since the only two patients with delayed-onset PTSD both had severe physical injuries. This has implications for the provision of support by professionals and others, and its influence in the cause and prognosis of PTSD. Since the population under study was of medico-legal referrals we are unfortunately unable to comment further on this.

Horowitz (1976) suggested that the symptoms of PTSD are phasic in nature and that delayed PTSD is an example of this pattern. Again in our results we can see this phasic pattern with two of the five individuals who were thought to have recovered from PTSD at six months having a re-emergence of symptoms at one year. It must be remembered when interpreting our findings at 12 months that this was in the anniversary week of the bombing, a time of intense emotional importance, of re-experiencing, and of increased media exposure, which may well be partly responsible for the re-emergence of symptoms.

The presence of the PTSD syndrome lends genuineness to a litigant but not exclusively so. Only half of the victims developed this particular syndrome. The other half also suffer psychological symptoms – fear, worry, depression, anger, etc. In seeking to help courts quantify 'nervous shock', some measure of duration, as well as quality, of symptoms suffered needs to be made, and ICD-9 codings still have some value in this respect.

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Psychiatric Morbidity in an Accident and Emergency Department
Characteristics of Patients at Presentation and One Month Follow-up

PAUL M. SALKOVSKIS, DAVID STORER, CHRIS ATHA and HILARY M. C. WARWICK

In order to evaluate how many patients presenting at accident and emergency (A&E) departments show signs of psychiatric disturbance, 140 consecutive *medical* presentations to an A&E department were evaluated using a range of simple self-report and rater measures, then followed up a month later. High levels of reported psychological problems were detected at screening, and these persisted at follow-up. Correlates of psychological disturbance and repeated attendance at A&E were investigated, indicating the relevance and feasibility of psychiatric intervention related to simple predictors.

With the important exception of psychiatric emergencies (Berrios, 1982; Dunn & Fernando, 1989), accident and emergency (A&E) departments have attracted little attention from psychiatric services. Research efforts in this area have concentrated on assessment and intervention in attempted suicide and other forms of self-harm (Hawton & Catalan, 1987). Knowledge of A&E attenders' psychiatric status is restricted to those patients referred to consultation/liaison services as psychiatric emergencies. Even among these patients, data tend to be restricted to retrospective studies (e.g. Fulop & Strain, 1986). There is a wide range of severity of the physical problems that present in this setting. A substantial proportion attend with minor physical ailments of no particular urgency and requiring little or no medical treatment.

Many A&E attendances may be accounted for by psychological factors. It has been argued elsewhere (Warwick & Salkovskis, 1985) that two separate but related processes can lead to consultation in medical settings. These are (a) the inconvenience, pain, and handicap caused by a complaint, and (b) worry engendered by the complaint, particularly fears concerning potential threat arising from *not* having it dealt with medically. Experience in liaison psychiatry supports this view, as does the effectiveness of placing a community psychiatric nurse (CPN) in an A&E department (Storer *et al*, 1987). Other indirect evidence comes from a study in which 'social difficulty' three to seven days after A&E attendance was evaluated by means of a telephone interview conducted by a social worker (Olsson *et al*, 1986). This study indicated that there was a problem requiring intervention (based on the 'felt need') in 38% of attenders.

It is therefore suggested that A&E attenders may be divided usefully into four principal categores: (a) those whose primary presentation is with overt psychological symptoms (psychiatric emergencies); (b) those presenting with disturbances of behaviour, often associated with psychiatric problems (e.g. self-poisoning, alcohol ingestion); (c) those presenting with physical ailments and for whom psychological factors strongly influenced attendance; and (d) those presenting with physical ailments for whom psychological factors played a lesser (or no) part in attendance. It is the third and fourth of these categories which are the focus of the present investigation.

There are three reasons for investigating this area. Firstly, if there is a large number of psychologically determined attendances, then this is likely to impair the efficiency of A&E departments. Secondly, although A&E staff are usually very aware of the psychological aspects of their work (Wright, 1986), it is unreasonable in present circumstances to expect them to be able to detect, assess, and manage all patients for whom the presentation is mainly psychological. Specialist training and back-up is required for such a task. It would therefore follow that, of necessity, many such patients do not receive the fullest appropriate treatment. Thirdly, given the present imbalance between levels of psychiatric morbidity in the community (Brown & Harris, 1978) and services available, the A&E department may well constitute a cost-effective way of accessing distressed people who would not otherwise come to the attention of psychiatric services.

As part of a larger programme designed to examine these and related issues, the study reported here included all non-psychiatric attenders at an A&E department physically able to complete screening instruments. The specific questions investigated were: (a) How does psychiatric morbidity (as measured by questionnaire) in this setting compare with that in other settings, such as general practice and community populations? (b) To what extent is disturbance detected at attendance in an A&E department a transient phenomenon confined to the reaction to a crisis such as physical injury? (c) Are 'cases' distinguishable from 'non-cases', and can any simple criteria be applied to make such a distinction more easily? (d) Is there a difference in 'disposal' of cases as opposed to non-cases? (e) What is the level of demand for treatment from patients, as indexed by requests for further help, at both presentation and follow-up?

Method

All patients attending the A&E department during three different times of the week and who fulfilled the selection criteria were screened. Times chosen were Saturday night (18.00–24.00), a weekday morning (07.00–12.00), and a weekday afternoon (12.00–18.00). Each occasion was sampled twice, giving six screenings in all. Selection criteria were: (a) age between 17 and 65; (b) no evidence of intoxication; (c) overt presentation not psychiatric; (d) physically able to complete questionnaires; (e) domicile within 50 miles of the hospital. In all, 140 patients were screened. Only one patient (a dental emergency) refused to comply.

Measures used were the 28-item General Health Questionnaire (GHQ-28; Goldberg & Hillier, 1979) and the Hospital Anxiety and Depression scale (HAD; Zigmond & Snaith, 1983). 'Caseness' thresholds used were those recommended by the authors. Demographic variables were taken from the usual casualty records, and patients were asked to fill out a short (10-item) scale (Health Care Questionnaire – HCQ) devised by the authors. This required yes/no answers to questions such as 'Have you attended your own doctor in the last month?' A subsample of patients were interviewed to check reliability of these items, which proved to be perfect. Medical staff treating the patient were asked to answer six questions and record the duration of their interview with the patient. The questions concerned the appropriateness of referral to a psychiatrist or community nurse, and whether attendance at casualty had been justified by the patient's physical condition.

At follow-up, the patients' state at that time was assessed by the GHQ-28 and HAD, and a further scale (Accident and Emergency Questionnaire – AEQ), similar to the HCQ in content but relating to the patient's present situation.

Patients were seen by a research nurse (CA) following reception by A&E nursing staff. The purpose of the study was explained to them, and they were handed the questionnaires together with a further explanatory note. Help in filling in the scales was offered as appropriate. If the volume of patients passing through the department became too great to deal with, every second arrival was screened in order not to bias the sample. In practice this happened infrequently. On the collection of the questionnaires, permission for follow-up was obtained. Medical staff were given their questions with the casualty card, and asked to fill this out at the same time. This was not always possible at busy times.

Follow-up was by post at one month. A reminder was sent two weeks later, and then a week after that. If no response was obtained at that point, patients were visited at home. A follow-up rate of 93% was obtained. Reasons for failed follow-up were: patient working away from the district and not contactable for the period of the study (two patients); moved from district and left no forwarding address (three patients); refused (two patients); not contactable (three patients).

Results

A total of 140 patients were screened (82 men, 58 women, mean age 30.04 years, s.d. 11.9).

The levels of psychiatric disturbance were relatively high in this sample, and were comparable to previously reported general practice samples, (which do not exclude patients attending for specifically psychiatric problems). Table I shows the levels of 'caseness' for the GHQ-28 and the two scales of the HAD, as well as the numbers who scored as 'cases' on at least one of these. The proportion of cases detected at the point of attendance and at follow-up is very similar, indicating that the total disturbance detected in this sample was persistent over one month. However, not all of this is accounted for by the same individuals. Analysis of 2×2 contingency tables indicated that a small number of patients cease to score in that range, while an approximately

TABLE I

Percentage scoring above threshold and the relationship between scores at presentation and follow-up

	GHQ-28		*HAD-D*		*HAD-A*		*All*	
	Presentation	*One month*	*Presentation*	*One month*	*Presentation*	*One month*	*Presentation*	*One month*
Percentage	26.5	25.5	8	12.5	28	34.5	36.5	38.5
χ^2	40.274*		37.54*		29.6*		32.2*	

*Presentation v. one month, $P<0.0001$.

HAD-A/GHQ $\chi^2=39.46$, $P<0.0001$, phi$=0.531$.

HAD-D/GHQ $\chi^2=18.84$, $P<0.0001$, phi$=0.367$.

HAD: Hospital Anxiety and Depression scale (A anxiety, D depression).

GHQ-28: General Health Questionnaire (scaled version).

TABLE II
Comparison of GHQ cases with non-cases (means (s.d.))

Variable	*Non-case*	*Case*
No. (male : female sex ratio)	103 (60 : 43)	37 (22 : 15)
Age: years	29.4 (11.1)	31.9 (13.8)
Duration of interview[1]	4.93 (4)	7.16 (6.4)*
Over-reacting[2]	0.21 (0.55)	0.63 (0.88)**
Referral to nurse[3]	0.09 (0.29)	0.37 (0.56)**
Referral to psychiatrist[4]	0.10 (0.41)	0.37 (0.56)**
Appropriate[5]	1.53 (0.66)	1.23 (0.59)*
Ratio trauma : minor injury[6]	4.8	1.27**
GHQ(a)	3.01 (2.2)	10.1 (4.6)***
GHQ(b)	2.62 (2.3)	9.54 (4.8)***
GHQ(c)	6.08 (1.9)	9.75 (3.8)***
GHQ(d)	0.85 (1.8)	4.40 (4.9)***
HAD-A	4.73 (2.9)	9.43 (4.9)***
HAD-D	1.89 (2.0)	5.24 (3.4)***

*$P<0.05$, **$P<0.01$, ***$P<0.0001$.

1. Time spent by A&E staff in initial interview with patient.
2. Rating (0–5) of staff judgement of how much the patient is over-reacting.
3. Rating of the extent to which the patient would benefit from CPN involvement.
4. Rating of the extent to which the patient would benefit from psychiatric referral.
5. Rating of how appropriate the attendance at A&E.
6. Ratio of minor injury to relative proportion of major trauma.

equal number become 'cases'. Unsurprisingly, the bulk of this movement is accounted for by patients scoring at or near threshold. Including those patients attending for overt psychiatric reasons raises the rate to just under 50%. Furthermore, the evidence is that the detected disturbance is not transient.

A comparison between the three different times of attendance revealed no significant differences. There had been an expectation of a greater proportion of cases on Saturday night, but this was not so. In fact, Saturday night tended to produce fewer cases. It is possible that this impression has arisen because of rates of admission of overdoses and alcohol-related problems.

There were no differences between cases and non-cases in the follow-up or disposal arrangements made by A&E.

Results from patients scoring above the GHQ threshold were compared with those of patients who did not score as cases (Table II). In particular, 'caseness' was significantly associated with minor rather than major trauma, despite which the nursing staff spent significantly more time with cases.

A&E staff questionnaire ratings of anxiety and depression did not differ between cases and non-cases; however, the ratings of judged need for further help (rated as given by either a CPN or a psychiatrist) were significantly different ($P<0.01$). The follow-up results showed an interesting pattern; the GHQ and HAD scores of the non-cases showed a small (non-significant) decrease. On the other hand, the scores of the cases showed a significant decrease in GHQ anxiety scale (paired $t=2.29$, $P<0.05$), while other scales showed a small, non-significant increase.

'Caseness' was significantly associated with a variety of other variables (Table II). One of particular note was the observation that 70% of cases had attended their general practitioner (GP) in the previous month, as compared with 29% of the non-cases ($\chi^2=19.2$, $P<0.001$). There was no association with expressed preference for hospital rather than own doctor, knowing what was wrong before A&E attendance, previous prescription of medication for 'nerves', and report of being overconcerned with health ($P>0.05$ in each instance).

At follow-up, the cases were significantly more likely than others to want further help with physical problems or illnesses (26% v. 64%, $\chi^2=12.35$, $P<0.001$) and with other worries/difficulties (19.5% v. 58.5%, $\chi^2=13.79$, $P<0.001$).

The data were analysed in an attempt to discover simple predictors of severe psychiatric disturbance. Items from the initial questionnaire which were related to GHQ and HAD scores were tested. Two items predicted well on this basis. Of 15 patients answering positively to the questions regarding difficulty coping with everyday life and whether they wished further help with their worries once they left hospital, 10 scored above threshold on the GHQ and 12 scored above threshold on the HAD anxiety scale. Thirteen of the 15 scored as cases on one or the other. In an attempt to determine the clinical relevance of these predictors, records were examined to evaluate how often these patients re-attended over the subsequent three months. These results were compared with those of a group of 15 age- and sex-matched controls (randomly chosen with respect to questionnaire results), and another group of 15 age- and sex-matched patients scoring above threshold for the GHQ. The results (Table III) indicate that the two questions predict re-attendance better than chance and better than the GHQ alone ($\chi^2=5.78$, $P<0.01$). Of the eight patients re-attending, five returned without any physical cause (including one panic attack and one overdose).

Thirty-two per cent of the patients said that they felt it would be useful for them to have someone from the hospital to discuss their worries after they went home. At follow-up this figure rose to 39%. Of this 39%, 87% wanted to discuss worries about their physical state, 75% wanted help with other worries or difficulties.

The comparison between the GHQ-28 and the HAD is of particular interest. This setting is a suitable one for such a comparison because the HAD was specifically designed to be used in the medical setting. Table IV shows the close relationship between the two scales. The HAD is distributed more normally in the depressed subscale than the comparable GHQ subscale (d) (cf. Aylard *et al*, 1987). The comparison indicates that the HAD has good concurrent validity in this setting; criterion validity

TABLE III
Rates of repeat attendance for patients selected using predictors compared with a matched control group and with unselected GHQ cases

Predictors	*Patients*	*Controls*	*GHQ cases*
No. returned	8	2	4
No. of return visits	19	3	6
Total in group	15	15	15

$\chi^2=5.785$, $p<0.01$.

TABLE IV

Correlations between GHQ and HAD subscales on each occasion

	Presentation						Follow-up						
	GHQ(b)	*GHQ(c)*	*GHQ(d)*	*GHQ(t)*	*HAD-D*	*HAD-A*	*GHQ(a)*	*GHQ(b)*	*GHQ(c)*	*GHQ(d)*	*GHQ(t)*	*HAD-D*	*HAD-A*
GHQ(a)	0.62	0.45	0.39	0.80	0.47	0.52	0.62	0.56	0.29	0.41	0.57	0.45	0.50
GHQ(b)	—	0.43	0.63	0.85	0.65	0.72	0.45	0.71	0.32	0.52	0.63	0.62	0.62
GHQ(c)	—	—	0.25	0.62	0.41	0.33	0.37	0.42	0.49	0.29	0.45	0.39	0.34
GHQ(d)	—	—	—	0.62	0.53	0.64	0.24	0.50	0.24	0.60	0.47	0.44	0.39
GHQ(t)	—	—	—	—	0.60	0.67	0.50	0.67	0.33	0.51	0.66	0.58	0.53
HAD-D	—	—	—	—	—	0.64	0.45	0.60	0.25	0.54	0.53	0.67	0.54
HAD-D	—	—	—	—	—	—	0.45	0.69	0.30	0.52	0.61	0.68	0.71

requires comparison of the two scales with a validated psychiatric interview. Towards this end, we have now completed a preliminary comparison (Atha *et al*, 1990*a*) using the Present State Examination.

Discussion

This study reveals relatively high rates of psychiatric disturbance in patients seen in an A&E department, consistent with those observed in other medical settings (Mayou & Hawton, 1986). The rates are at least as high as in studies of general practice where psychiatric reasons for attendance were not used as exclusion criteria (Stanley & Gibson, 1986). Patients attending GP clinics are commonly attending because of psychiatric problems. A more appropriate comparison for an A&E sample would be with a group taken from a non-clinic community setting. The figures of 'caseness' in this type of population are 11.3% (Goldberg *et al*, 1976). Furthermore, we have subsequently replicated the rates found in the present sample (Atha *et al*, 1990*a*).

It is not possible to account for these results in terms of elevated levels of state anxiety resulting from being in a hospital setting at the time the questionnaires were filled out, as the rates remain the same at follow-up over a month later, even though anxiety did specifically decline in the identified cases. On the other hand, the results from the HAD appear to suggest that anxiety and depression both increase slightly in the intervening period. An alternative possibility concerns the longer-term effects of physical injury as a life event. There is evidence (Paykel, 1982) that such life events are followed by an increased incidence of psychiatric disturbance, particularly depression. However, this work usually refers to major life events with substantial impact. Most of the injuries sustained by the patients in this study (particularly the identified cases) were minor, sometimes requiring rapid attention but without longer-term sequelae.

Another possibility is that the choice of hospital as medical facility to attend is at least partly determined by the psychological state of the patient; thus, a worried or depressed patient is more likely to go to hospital than one who is not. A further determinant may be the reception that the patient expects to receive from the GP. We note that the cases reported significantly more GP attendances in the month before A&E attendance, but did not express a preference for hospital attendance.

In conclusion, we found that psychological problems as detected by questionnaire were common in the A&E department studied, and that the disturbance noted was persistent. Despite recent evidence supporting the general validity of the questionnaires used (Aylard *et al*, 1987), the extent to which the measures used reflect psychiatric illness requires further validation, currently being undertaken. However, the principal intention in this study was not to make a definitive statement about the prevalence of psychiatric problems, but rather to examine the relationship between self-reported problems and variables relevant to the efficient management of patients in the A&E department. We found that a group of more severely disturbed patients can be detected on the basis of two yes/no questions: are you coping with everyday life, and do you wish further help with your worries once you leave hospital? These questions also predict *a priori* repeated attendance in the A&E department. This was true in the present sample, in the subsequent replication sample (Atha *et al*, 1990*a*), and in the study in which such patients were treated as part of a controlled trial (Atha *et al*, 1990*b*). Staff show some awareness of the problems presented by those identified as cases, reflected in the ratings of need for further help and the greater time spent with cases, despite the fact that cases were less likely to show severe trauma.

These findings suggest that greater psychological input to A&E departments would be useful. Such input would free A&E staff to deal more efficiently with physical trauma, while also serving the needs of those patients in whom attendance relates to psychological factors. Our previous work (Storer *et al*, 1987) raised the possibility that a CPN attached to A&E departments but working with patients in the community may fulfil the requirements. In two recently completed controlled trials, we have been able to confirm the effectiveness of a time-limited problem-solving approach (c.f. Hawton & Kirk, 1989) in patients who repeatedly attempt suicide (Salkovskis *et al*, 1990), and in those identified as repeat attenders using the predictor criteria derived from the present study (Atha *et al*, 1990*b*).

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Patients Leaving Hospital Without the Knowledge or Permission of Staff – Absconding

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A series of 100 consecutive psychiatric in-patients who absconded from a psychiatric hospital a total of 231 times over ten months was studied. The reasons given for absconding, duration of absence, and mode of return are described. The legal status and ethnic origins of absconders differed significantly from those of the base hospital population.

The open-door policy introduced into British mental hospitals, and the Mental Health Act 1959, led to the removal of many of the restrictions on patients' liberty that were part of the era of locked wards and padded cells. The increase in patients' freedom has created the problem of some patients leaving hospital before it is desirable. Such patients often generate a great deal of concern among members of staff, relatives, and the community at large, and may be a danger to themselves or others.

This problem was recognised by Muller (1962), who studied a series of 98 absconders from Powick Hospital and interviewed the 73 patients (75%) who returned. Muller found that 50% of absconders left on more than one occasion, and believed there was generally one main reason for a patient absconding. Antebi (1967) reviewed the case notes of 166 absconders and found 71% were informal at the time they absconded, and were as likely to escape from locked wards as open wards. He expressed concern that 39% of male absconders had a criminal record, and that a third of males who did not return were detained under section 60 or 65 of the Mental Health Act 1959 (court orders). However, both studies were carried out over 20 years ago, and not only has psychiatric practice and treatment changed considerably since then, but the Mental Health Act 1983 has also been implemented. Apart from a study by Tomison (1989), the only other studies were carried out in the United States over 15 years ago. In view of this it was decided to study a new series of absconding patients in order to examine the extent of the problem, outline the patients' characteristics, and determine the outcome of absconding and its management.

Method

Springfield Hospital is a large Victorian psychiatric hospital serving a catchment-area population of 355 000 in the London boroughs of Merton and Wandsworth. There are 670 beds, of which 180 are acute general adult psychiatric beds and 20 are on locked wards. Any patient who is absent without the permission of staff and is considered to be frail, severely depressed, suicidal, a cause for serious concern, or has exhibited recent disturbed behaviour is treated as an absconder. This involves searching the hospital and informing relatives and the police.

Comprehensive data were gathered on 100 consecutive absconders, including demographic details, psychiatric history, reasons for absconding, and outcome by examining patients' case notes and interviewing staff involved. Patients were assigned to an ethnic group on the basis of their own and Staff's view as to which group was most appropriate. One of the investigators (JF) interviewed patients on their return using a semistructured interview.

In addition to the prospective study, the fate of 87 consecutive patients who had absconded between one year and 18 months previously was studied retrospectively by contacting general practitioners, coroners, and examining case notes.

Results

The 100 patients absconded a total of 231 times between October 1987 and August 1988. Successful interviews were carried out with 76% of absconders on their return. Ten per cent of patients failed to return, 4% were unwilling to co-operate, 3% had been discharged before they were interviewed, and 7% were unable to answer reliably, as they were demented. There were more male absconders (61%), and they tended to be slightly older (mean age for males = 37.5, s.d. = 17.6 years, mean age for females = 36.5, s.d. = 12.8 years, difference not significant).

The absconding group differed from the hospital population only in their status and ethnic origin. Most absconders (70%) were subject to a section of the Mental Health Act 1983 at the time they absconded, and they were more likely to have been admitted under a section of the 1983 Act (Table I).

Patients of UK/Irish origin were under-represented, while all the other ethnic groups were over represented (Table II). The ethnic background of patients also affected their status at the time of absconding, with almost all the

TABLE I

Status of absconders compared with the hospital population at the time of admission

Status	*Absconders (%)*	*Population (%)*
Informal	51	81*
Section 2 (for assessment)	14	6.4
Section 3 (for treatment)	2	1.4
Section 4 (emergency admission)	10	3.0
Section 37 (court order)	5	0.7
Section 37/41 (court order with restrictions)	1	0.1
Section 136 (police order)	17	7.4

*Informal v. formal, Yates' $\chi^2=20.05$, d.f. = 1, $P<0.001$.

TABLE II

Ethnic groups of absconders and in-patients

Ethnic group	*Absconders (%)*	*In-patients (%)*
Afro-Caribbean	26	16
African	8	3
UK/Irish	49	66
Other European	10	6
Asian	6	5
Other	1	4

Afro-Caribbean patients who absconded being formally detained (Table III).

Using the ICD-9 classification (World Health Organization, 1978), the single largest group of absconders was suffering from schizophrenia (45%), followed by manic-depressive psychoses (17%), paranoid psychoses (9%), personality disorders (8%), neurotic disorders (5%), and organic psychoses (7%), with other diagnoses accounting for the remainder.

Eighty per cent of absconders had had previous admissions to psychiatric hospitals, and 49% had absconded during previous admissions.

There were 39 patients who absconded on more than one occasion during the study. They absconded between 2 and 12 times (mean = 6.8, s.d. = 4.1). The majority of patients who repeatedly reabsconded (77%) were compulsorily admitted under a section of the Mental Health Act (32% under section 136, police admissions) and were compulsorily detained at the time of absconding. There were large differences between the ethnic groups: only 20.4% of patients of UK/Irish origin reabsconded compared with 64% of patients of Afro-Caribbean origin (Table III).

The mean duration of absence from hospital was 23.1 hours (s.d. = 34 hours), with 40% returning within 12 hours and 10% failing to return. Of those who did return, surprisingly 35% returned on their own, police returned 39%, and friends or relatives 8%.

Patients expressed a variety of feelings on their return to hospital, which were often related to their mode of return. Many of the patients brought back by the police were angry, and overall 24% of absconders were angry about being back in hospital. A further 19% of patients were unhappy about being in hospital. Twelve per cent of patients were relieved at being back. This was particularly common among those who returned themselves. Twenty per cent of patients were intending to stay in hospital after their return, while 18% admitted that they might well abscond again at some point.

Staff were concerned when patients absconded and were relieved on their return. They tended to be less concerned about patients who had absconded often in the past, and in some instances felt frustrated when patients repeatedly absconded.

Various reasons were given as being important factors in patients' decisions to abscond. A number were concerned with the hospital, including the stigma of being in a psychiatric hospital (17%), and disliking the staff (13%), the food (11%), the ward (8%), and the lack of privacy (7%). Nineteen per cent complained of being disturbed by other patients. In addition, worry about their homes (18%) or families (10%) were important factors. A few patients left because of abnormal beliefs, including fears that they might be harmed in some way in hospital (19%). Others left in response to abnormal experiences, such as command

TABLE III

Status of absconders, and numbers of reabsconders, by ethnic group

Ethnic group	*Informal*	*Formal*	*Absconded once*	*Absconded more than once*
Afro-Caribbean	0	25*	9	16
African	2	6	4	4
UK/Irish	25	24*	39	10
Other European	4	6	5	5
Asian	1	5	2	4
Other	0	1	1	0

*Afro-Caribbean compared with UK/Irish, Yates' $\chi^2=16.69$, d.f 1, $P<0.001$.

hallucinations (5%). One patient left with the intent of committing suicide, and one patient committed suicide.

When patients absconded 48% went home, and 19% went to another specific place, while 16% wandered aimlessly.

There were no changes made in the management of the wards when patients absconded, but patients were often observed more closely on their return. Although there was no set policy for managing returned absconders, those who continued to give rise to concern were rapidly assessed. Seventy per cent of the patients returned by the police had changes made in their management. Twelve per cent of absconders were transferred to the locked ward on their return. This was more likely if they had absconded previously. Changes were made in medication in 11%. The status of a number of patients was changed. Some were considered well enough to be discharged (6%), some were well enough to be allowed to go on leave (3%), and 2% were sufficiently ill to be compulsorily detained in hospital under section 5(2). There were no changes made in the management of 56% of patients.

Of the 87 earlier absconders followed up, 79 were still known to the psychiatric services or their general practitioners, while eight patients were unaccounted for, with no information available as to their health or whereabouts Six patients had committed suicide within 18 months of their absconding.

Discussion

This study was designed to outline the characteristics of absconders from a British psychiatric hospital. It is the only prospective study to date in which patients have been interviewed upon their return. There were no significant differences between the absconders and hospital population of patients who did not abscond, except in their legal status and ethnic origin. The finding that more patients who abscond have been admitted compulsorily and were formally detained is consistent with previous studies (Muller, 1962; Antebi, 1967; Tomison, 1989). However, differences in the rates at which ethnic groups absconded are more difficult to interpret, and have not been previously reported. Afro-Caribbean patients who abscond were almost always formally detained, and they were also far more likely to reabscond. There is a need to address these issues, as they may have important implications for current practice.

The characteristics of patients absconding from Springfield Hospital in London are very similar to those of patients absconding from Barrow Hospital in Bristol (Tomison, 1989). Despite the changes in psychiatric practice and the implementation of the Mental Health Act 1983, it is surprising how the results of the present study and Tomison's (1989) differ little from Muller's and Antebi's studies over 20 years ago. The present series is similar not only in terms of their demographic details, diagnoses, and duration of absence, but also their legal status. The proportion of patients who repeatedly absconded is similar, but more patients in the present study went home (48%) than in Antebi's study (28%), and more returned to hospital on their own. The proportion of patients absconding appears to have remained essentially unchanged, although far fewer are now kept on locked wards. Muller found there was one reason for absconding in most cases, and listed treatment failure (often schizophrenic patients responding to command hallucinations), alcoholism, and being influenced by other patients as the most common reasons. These factors were reported rarely to account for absconding in the present study, and rarely was a single reason given.

Although many patients returned to hospital on their own after a relatively brief absence, there was one suicide during the study. The retrospective finding of suicide in six patients within 18 months of absconding warrants further examination, as absconding during treatment may well be an underestimated risk factor in suicide. The finding that in many instances few if any changes were made in patients' management on their return warrants further investigation. Whether more needs to be done for patients who abscond and whether staff treat some 'absences' with too much importance needs to be determined. This is important, as a prompt response from agencies outside the hospital including the police is more likely if fewer patient absences are treated as absconding.

Patients gave a variety of reasons as important in their decision to abscond, such as complaints about the food and lack of privacy, which could be ameliorated by additional resources.

The continuing importance of absconding from mental hospitals has been demonstrated and a number of important aspects identified. Among these is the need for further research in this area.

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Affect, Affective Disorder and Schizophrenia
A Neuropsychological Investigation of Right Hemisphere Function

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Performance on a happy–sad chimeric face test was used to examine the role of right hemisphere activation in positive and negative affect, both normal and abnormal, as well as in schizophrenia. This test is known to elicit a left-sided perceptual bias in right-handed normal subjects. Happy and sad mood in normals did not influence the perceptual bias. Depression and mania were associated with reduced and increased biases respectively, while schizophrenics showed no bias to either side. Possible explanations are right hemisphere hyperfunction in mania, moderate relative hypofunction in depression, and severe relative hypofunction in schizophrenia. The marked difference between mania and schizophrenia supports distinct pathophysiologies underlying the two conditions.

The distinction between depressed mood as it occurs in normal people and pathological depression which constitutes an illness has long been the subject of debate (Gilbert, 1984). A slightly less ancient controversy surrounds the issue of whether psychosis is a unitary phenomenon or whether schizophrenia can be distinguished from affective psychosis (Kraepelin, 1921). The two contrasting views on the question of mood disturbance have been described as the 'Hippocratic' or dimensional view (Kendell, 1976) and the 'Platonic' or categorical view (Akiskal, 1987). The former holds that there exists a spectrum of affect from pathological sadness at one end to pathological elation at the other, with normal variations of mood situated somewhere in the middle. The latter view is that relatively clear distinctions can be made between normality and abnormality as well as between subtypes of depression, depending on such factors as the propensity to develop mania, the presence of delusions, response to treatment, etc. These contrasting viewpoints also apply to arguments as to whether the most severe mental illnesses lie on a spectrum, from affective, through schizoaffective, to the schizophrenic psychoses (Kendell & Gourlay, 1970; Crow, 1986), or whether schizophrenia, mania and depression are distinct disease entities (Kraepelin, 1921).

Neuropsychological and psychophysiological research has not confirmed a categorical difference between normal and abnormal mood states, but it has uncovered different contributions to mood from the two hemispheres (Flor-Henry, 1986). Some research points to a link between affective disorder in general and dysfunction of the right hemisphere (RH) (Taylor & Abrams, 1987) while other work looking at the effects of known brain lesions suggests that mania and depression are the result of dysfunction of the left and right hemisphere (Sackeim *et al*, 1982) or vice versa (Robinson *et al*, 1988). This confusion is mirrored in the research on normal subjects (Silberman & Weingertner, 1986). For example, electroencephalographic (EEG) measures of arousal are increased over the RH during depressed mood (Tucker *et al*, 1981) and over the left hemisphere (LH) during happy mood (Davidson *et al*, 1979; Ahern & Schwartz, 1985). Some studies of affect recognition using divided visual fields have shown an RH advantage in perceiving sad facial expressions and an LH advantage for happy expressions (Reuter-Lorenz *et al*, 1983; Nalate *et al*, 1983) while other studies have shown an RH advantage regardless of the valence of the affect in both normal (Ley & Bryden, 1979; Strauss & Moscovitch, 1981) and brain-damaged populations (Borod *et al*, 1986). Much of this work assumes that normal variations in mood and the affective disorders share the same underlying mechanisms, a view which has not received unanimous support in the literature (Davidson *et al*, 1985).

Regarding the psychoses, Johnstone *et al* (1988) showed that treatment response could not be used to validate separate diagnoses of schizophrenia and manic–depressive psychosis. Psychophysiological research on smooth-pursuit eye movements and evoked potentials (Holzman, 1987) has been more successful in showing differences. Most evidence from structural brain damage points to abnormalities in the LH in schizophrenia only (Brown *et al*, 1986). Nevertheless, some investigators have found abnormalities of the corpus callosum (Rossi *et al*, 1988; David, 1987), and others have identified functional disturbances attributable to the RH (Bellini *et al*, 1988; Cutting, 1985). Recognition of facial affect in particular has been found to be impaired in some schizophrenic patients (Morrison *et al*, 1988) and has been attributed to deficient information processing in the RH (Magaro & Chamrad, 1983).

In order to clarify some of these issues, particularly the contribution of lateralised hemisphere dysfunction, a test is needed which is known to reflect RH function and which can be easily administered to normal subjects during different moods as well as to psychiatric patients. One test which reliably elicits an RH advantage and does not require specialised testing equipment is the free-vision chimeric faces test (Campbell, 1979; Levy *et al*, 1983). Levy *et al*'s test requires the subject to compare two composite facial photographs to determine which is the happier. The first face comprises one half bearing a neutral expression and the other half smiling. The comparison picture is the mirror image of the first. Normal right-handed subjects show a consistent bias towards the composite whose smiling half-face falls to *their* left: a 'left hemifacial (LHF) bias'. A modified version of this test has recently been developed using happy–sad chimeric face drawings (David, 1989; see also Jaynes, 1976). When right-handers are required to judge whether the face is happy or sad a strong LHF bias effect is again observed. (There is also a tendency to rate the faces as sad more often than happy but this 'sad bias' is independent of the LHF bias.) There is evidence that the LHF bias is due to RH 'dominance' in spatial organisation (Grega *et al*, 1988) and is robust enough to withstand manipulations of attention (Schwartz & Smith, 1980) and processing strategy (Gilbert & Bakan, 1973; Bennett *et al*, 1987). Other evidence supporting the RH's role in this phenomenon includes the observation that left-handed subjects do not show a consistent bias to either side (Lawson, 1978; Levy *et al*, 1983; David, 1989). Also, studies on patients with focal brain resections (Kolb *et al*, 1983) have revealed that LH lesions do not alter the bias whereas right posterior lesions do.

The effect of mood was studied by David (1990) using happy–sad chimeric faces. Depression and elation in normal volunteers, induced by the Velten procedure (Velten, 1967; Teasdale & Fogarty, 1979), resulted in substantial effects on happy and sad choices respectively but did not affect the LHF bias. Jaeger *et al* (1987) gave depressed patients Levy *et al*'s test and found that the normal LHF bias was attenuated, supporting RH dysfunction. A relationship between severity of depression and LHF bias was not reported. There is therefore a hint that depressive illness and depressed mood can be distinguished with respect to performance on the chimeric-face test. The present study used happy–sad chimeras since, unlike those employed by Levy *et al* (1983), they require both happy and sad choices while allowing each of these to be rated in terms of left or right bias.

Method

Twenty-five in-patients with major depression (two of whom had had questionable hypomanic episodes in the past), 15 with mania or hypomania, and 20 with schizophrenia according to DSM–III criteria (American Psychiatric Association, 1980) were tested (Table I). Patients with schizoaffective disorder were not included. All were classified as purely right-handed (Annett, 1970). Sixty normal right-handed subjects drawn from hospital staff were used as controls. A further ten normal volunteers participated in the mood-induction procedure.

Subjects rated their mood at the time of testing on a visual analogue scale. The depressives were also scored on the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967). The happy–sad chimeric faces test was given to all subjects and required them to make affective judgements on 48 faces (12 original faces and 12 mirror images, each face presented twice). That is, they were asked to say if each face, viewed in turn in free vision from a booklet (available from ASD on request), looked happy or sad (Fig. 1). All clinical subjects demonstrated their understanding of the text by correctly classifying the facial expressions of 8 non-chimeric face drawings. If they were not sure, they were encouraged to guess. Testing took approximately 15 minutes. Left bias was scored by subtracting the number of responses which coincided with the half-face to the (viewer's) left (min. = 0, max. = 48) from those which corresponded to the half-face to the right (min. = 0, max. = 48). The possible range for left-bias scores was therefore +48 to −48.

Results

Table I shows the mean left-bias scores for individual groups. Normals and manics showed significant left bias (binomial test and one-sample *t*-test). Depressives showed a weaker bias which reached significance only on the one-sample *t*-test. Schizophrenics showed no significant

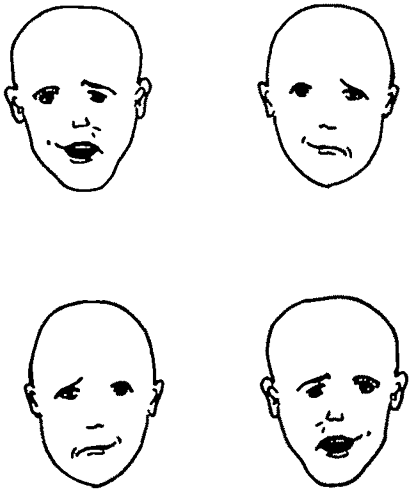

FIG. 1. Happy–sad chimeric faces: original and mirror image. Examples with open- and closed-mouth expressions.

TABLE I
Comparison of psychiatric patients with normal controls

	Normals (n = 60)	*Depressives* (n = 25)	*Manics* (n = 15)	*Schizphrenics* (n = 20)
Mean age (s.d.) years	33.4 (10)	42.2 (16.4)	36.7 (12.9)	35.5 (13.0)
Sex				
female	30	15	7	8
male	30	10	8	12
Mood[1]: mean (s.d.)	53.1 (14.7)	26.7 (18.8)	73.7 (24.2)	55.4 (22.8)
Left bias[2]				
No. of subjects scoring:				
>0	49	16	15	10
<0	7	7	0	4
=0	4	2	0	6
Binomial test	$P<0.0001$	$P=0.09$	$P<0.0001$	$P=0.18$
One-sample *t*-test	$P<0.0001$	$P<0.01$	$P<0.0001$	$P=0.4$
Mean (95% CI)	9.8 (7.5–12.1)	6.3 (2.8– 9.9)	12.9 (9.2–16.6)	1.1 (−1.2– 3.4)
Sad bias[3]				
Mean (95% CI)	7.2 (4.5– 9.8)	5.1 (0.0–10.2)	2.7 (−7.2–12.6)	3.2 (−3.9–10.3)

1. Visual analogue scale, range 1–100. 1 = saddest ever, 100 = happiest ever.
2. Left bias equals the number of responses which correspond to the left hemiface minus the number which respond to the right hemiface.
3 Sad bias equals the number of sad responses minus the number of happy responses.

bias in either direction, regardless of statistical method.

Analysis of variance showed that there were main effects by diagnosis: for left bias (d.f. = 3,116, $F=7.96$; $P<0.001$); for mood ($F=25.0$; $P<0.001$); and for age. ($F=3.2$; $P<0.05$); but not for sad bias ($F=0.7$; $P=0.5$). There were no sex-by-diagnosis interactions. Examining left bias by applying *post-hoc* *t*-tests (two-tailed) showed that manics differed significantly from depressives ($P=0.01$) but neither differed significantly from normals ($P=0.1$). Schizophrenics differed significantly from depressives ($P<0.02$), manics ($P<0.001$), and normals ($P<0.001$). Manics showed a greater LHF bias, and depressives and schizophrenics showed a reduced bias with respect to normals. These results contrast with the striking lack of effect in either direction when either elated or depressed mood was induced in normals (Fig. 2).

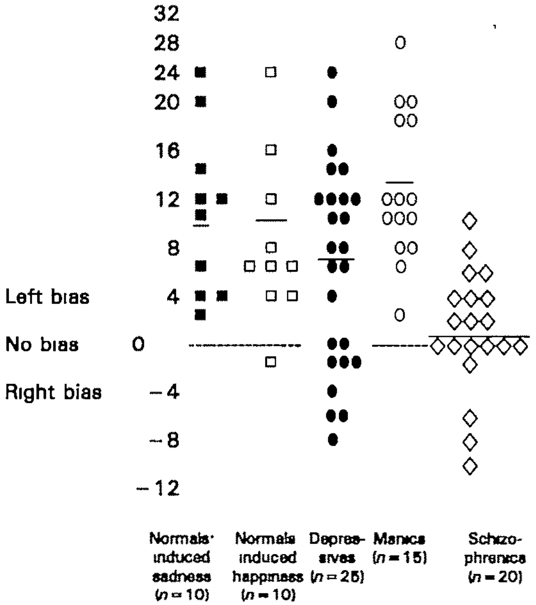

FIG. 2. Scatter plot showing left-bias scores (each point indicates one subject's score; bars show means for each group).

The depressives were slightly older than the other groups but since there was no correlation between age and left bias in either the normals or the affective groups this is unlikely to account for the differences. There was a positive relationship between age and left bias in the schizophrenic group ($r=0.42$; $P<0.05$). This is unlikely to have detracted from the significant comparison with the manic since the groups were of similar ages.

All groups, on average, displayed a positive sad bias (a tendency to say sad more often than happy), the magnitude of which did not differ significantly between them, although the extent of the sad bias in the manic patients was predictably little greater than zero. Sad bias did not correlate with left bias for any group. Within the 60 normal subjects (not shown in Fig. 2), the 10 subjects in the mood induction group, the schizophrenics and the manics, there were no correlations between self-rated mood and left bias. In the pathologically depressed group, however, there was a significant negative correlation (Pearson's $r=-0.46$; $P=0.01$). That is, as sadness increased so the left bias decreased, although their HRSD scores (mean = 23.48, range 16–31) did not correlate with left bias ($r=0.03$).

Discussion

The results point to different cerebral mechanisms between schizophrenic and affective disorders on the one hand and between normal and pathological mood states on the other. Normal subjects experiencing variations in mood do not exhibit any change in LHF bias, a reliable RH phenomenon (Grega *et al*, 1988; David, 1989). These data confirm a prior report of reduced LHF bias in clinically depressed patients (Jaeger *et al*, 1987) and point to RH hypofunction in depression, although in this study, the reduction compared with that of normal controls did not quite reach significance. In addition, hypomanic and manic patients showed an increased LHF bias, suggesting RH hyperfunction. The schizophrenic group showed no significant bias, pointing to RH hypofunction (Magaro & Chamrad, 1983; Cutting, 1985). This indicates that schizophrenia is quite unlike mania in this respect.

It could be argued that the results only support relative dysfunction of one hemisphere in relation to the other. However, LHF (RH) bias was not enhanced following left-sided cortical excisions (Kolb *et al*, 1983). Thus, LH hypofunction is an unlikely explanation for the exaggerated LHF bias displayed by the manic group. However, since there is no satisfactory neurological model of hyperfunction, it is not yet possible to say with certainty whether the schizophrenics' performance, and to a lesser extent the depressives', is a consequence of RH hypo- or LH hyperactivity or a combination of both.

Cerebral blood flow studies provide support for an imbalance of hemispheric function in both depression (Uytdenhoef *et al*, 1983) and schizophrenia (Gur *et al*, 1985). This is further complicated by the acknowledgement that any such hemispheric imbalance is likely to involve the corpus callosum. A functional interhemispheric disconnection might be predicted to increase right hemifacial (LH) choices and hence reduce the left bias, given that vocal (LH) responses were required (Levy *et al*, 1972), as in this study. However, callosal disconnection might be expected to produce the opposite effect, if manual (particularly left-handed) responses were asked for. The results for the schizophrenic group therefore are not incompatible with an abnormal corpus callosum, but replication using manual responses would be required to confirm this.

The question of whether the abnormalities postulated reflect a 'state' or 'trait' cannot be answered without re-testing the patients after recovery or testing manic patients during a depressive phase of their illness. The latter strategy has been utilised in a single case study using EEGs which showed LH and RH hyperactivation respectively during depressive and manic episodes (Sackeim & Decina, 1983), which is consistent with the data reported here. The fact that the HRSD score did not correlate with left bias, although self-rated mood did, means that no firm conclusions can be drawn as to the effect of the severity of a depressive illness. The pattern of results obtained indicates that within a depressive illness, subjective negative mood reduces a weakened LHF bias and this might be construed as providing support for the notion that the altered bias reflects a depressive 'state'. Another factor to be taken into account is medication. All but two schizophrenics and all but three manics were on neuroleptics and all but four depressives were on antidepressants. Jaeger *et al* (1987) found that the administration of tricyclic antidepressants did not affect the bias in their depressives. Furthermore, the fact that manic and schizophrenic groups differed so widely in their biases despite being treated with neuroleptics is good evidence that there is no confounding drug effect.

In conclusion, differences of a qualitative kind were demonstrated between normal and abnormal mood states and especially between affective and schizophrenic disorders using a simple test sensitive to RH functioning. It should be added that the cerebral basis for the test performances described is a matter of inference and that parallel studies which measure metabolic or electrophysiological activity directly are needed before such a basis can be certain. Nevertheless, the test is eminently suitable for research with psychiatric groups and other clinical populations. Applying the technique to 'schizomanic' patients (Brockington *et al*, 1980) to see whether they follow the pattern of pure manics or schizophrenics would be a critical experiment. Devising a task which produces a reliable LH (right spatial) bias would be most useful in further clarifying the nature of the dysfunction reported here, although so far no such task is available.

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The Prevalence and Diagnostic Significance of Schneiderian First-Rank Symptoms in a Random Sample of Acute Psychiatric In-patients

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Ninety-nine acute in-patients were interviewed within four days of admission to hospital. The interview included items concerning all 11 first-rank symptoms. Two definitions (wide and narrow) of each first-rank symptom were employed. Three definitions of schizophrenia and a research diagnosis of major affective disorder were used to determine their relationship with first-rank symptoms. Of subjects with schizophrenia, 73% had first-rank symptoms, as had five (14%) of 34 subjects with affective disorder. The results do not support the hypothesis that first-rank symptoms are specific to schizophrenia, although the specificity of first-rank symptoms for schizophrenia increased if a narrow rather than a wide definition of symptoms was employed.

Since the original division of the functional psychoses into manic–depressive illness and schizophrenia, clinicians have sought a reliable method by which to distinguish them. Kurt Schneider (1959) considered that the two psychoses could be distinguished by reference to 11 'first-rank' symptoms. He stated that in the absence of an organic psychosis, first-rank symptoms were characteristic of schizophrenia, although he added that they were in no way fundamental to that disorder and that schizophrenia could be diagnosed in their absence. Mellor (1970) showed that they occur frequently enough in a patient population diagnosed as schizophrenic to be useful, but the claim that they occur only in schizophrenia has come under critical scrutiny in the past two decades. In a study of mania, Taylor & Abrams (1973) found that 12% of their sample had first-rank symptoms. Using data from the International Pilot Study of Schizophrenia and a research group of sufferers from affective disorder, Carpenter *et al* (1973*b*) found that 23% of those with an affective disorder had first-rank symptoms, as did certain patients with neurosis and personality disorder. Brockington & Leff (1978) have shown that first-rank symptoms do not predict a poor outcome if schizophrenia is thought to have a poor prognosis anyway. These and other studies (Carpenter & Strauss, 1974; Koehler *et al*, 1977; Koehler & Seminario, 1978) have gradually eroded the status of first-rank symptoms as pathognomonic of schizophrenia.

The impetus for this study has come from two sources. Wing & Nixon (1975) in a critique of the studies of Carpenter *et al* (1973*b*; Carpenter & Strauss, 1974) suggested that the results of studies reporting first-rank symptoms in disorders other than schizophrenia may have depended upon the definition of first-rank symptoms used, and more particularly on how narrowly they were defined. Koehler (1979) has examined currently available definitions of first-rank symptoms and demonstrated that they are defined differently by different authors. He showed that each first-rank symptom could be viewed as a continuum between a narrow and a wide definition. These studies suggested that it might be useful to re-examine the specificity of first-rank symptoms for schizophrenia using narrow and wide definitions. To my knowledge, such a study has not previously been reported.

Most previous studies have used selected samples rather than a general sample of psychiatrically ill patients. This study was designed to find the frequencies of broadly and narrowly defined first-rank symptoms in a sample of acute mental hospital admissions and find how they relate to diagnosis. The main hypothesis was that the use of narrowly defined first-rank symptoms would produce greater specificity for schizophrenia.

Method

Subjects were interviewed on one day each week over a year, excluding breaks for holidays. The sample reflected admissions over one complete year.

Patients admitted to the acute admission wards of St Nicholas' Hospital, Newcastle upon Tyne, were interviewed, excluding the psychogeriatric admission wards. Patients were excluded if profoundly deaf or unable to speak English. To be included in the study, patients had to be in hospital for a minimum of four days, which allowed the researcher to interview the subject on more than one occasion if necessary. In practice, no patient was excluded by staying less than four days. All admissions over the 24 hours of the chosen research day were interviewed.

A semistructured interview schedule of known reliability, the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978) was used to interview each subject, with the addition of a first-rank symptom questionnaire. Each subject was then assigned a diagnosis using the Research Diagnostic Criteria (RDC: Spitzer *et al*, 1978), Carpenter's Flexible System (Carpenter *et al*, 1973*a*), and the New Haven Index (Astrachan *et al*, 1972). The SADS was used as it is specifically structured to provide RDC diagnoses. Carpenter's Flexible System and the New Haven Index were used as they do not heavily rely on first-rank symptoms to define schizophrenia. All symptom-based definitions of schizophrenia make use of first-rank symptoms and it follows that first-rank symptoms could not be tested against a diagnostic system independent of first-rank symptoms. To overcome this problem the number of definitions satisfied by each subject is correlated with the presence of narrowly and widely defined first-rank symptoms.

The symptom questionnaire contained questions on all 11 first-rank symptoms. Each symptom was rated using the dichotomy of wide versus narrow definitions proposed by Koehler (1979). The various definitions were drawn from Mellor (1970), Fish (1967), Wing *et al* (1974), and Taylor & Heissler (1971). The narrow definition was in all cases that of Mellor (1970). The distinguishing features used to differentiate narrow from wide first-rank symptoms were:

(a) made feelings, made impulses, made actions, somatic passivity
  (i) narrow: the subject experiences these sensations as not being his own but as arising from an outside source
  (ii) wide: the subject experiences them as his *own*, but as being controlled from outside
(b) thought insertion
  (i) narrow: the subject experiences thoughts as not being his own and being inserted into his mind
  (ii) wide: the subject experiences them as being controlled from outside
(c) thought withdrawal
  (i) narrow: the thoughts cease in the subject's mind owing to the direct influence of an outside agency
  (ii) wide: thought block is accepted in the absence of an experience or thoughts being interfered with by an outside agency
(d) thought broadcast
  (i) narrow: the subject not only experiences thoughts leaving the confines of his own mind, but also experiences them being shared with others
  (ii) wide: either thoughts leave the confines of the subject's mind but are not shared or they are so loud that others can hear them
(e) voices in discussion, voice commentary, audible thoughts
  (i) narrow: the hallucination is heard outside the subject's head (true hallucination)
  (ii) wide: The voices are heard only inside the subject's head/mind
(f) delusional perception
  (i) narrow: the delusional idea and the perception are directly linked to one another such that the delusional idea cannot be separated from the perception and occurs in very close temporal relationship to it
  (ii) wide: there is a relatively loose link between a perception and a delusional idea and the delusional idea is often linked with other phenomena

Results

Of 109 patients potentially available for inclusion, three refused to be interviewed and three interviews were not suitable for research purposes. In these six cases there was no evidence of first-rank symptoms from interview or scrutiny of case notes. Four patients had organic brain disease and are not included in the main sample as they were not suitable for interview using the SADS. However, they were interviewed for the presence of first-rank symptoms, and are reported separately. The remaining 99 subjects comprised the main sample.

The age range of the 99 subjects (59 women, 40 men) was 18–89 years (mean 40.6); 38 were single, 35 married, and 26 separated or widowed. There were 10 first admissions, of whom one had first-rank symptoms.

Sixteen subjects had first-rank symptoms (mean per subject 2.6, median 2, range 1–9), only three had wide first-rank symptoms in isolation, and two had first-rank symptoms according to both definitions.

The most common symptom was voices in discussion, eight subjects having this symptom. No subject had a delusional perception. Otherwise made actions was the least common, with the others more or less evenly distributed.

First-rank symptoms and diagnosis

Table I shows the relationship between RDC diagnosis and first-rank symptoms. The majority of those with first-rank

TABLE I
RDC diagnosis and first-rank symptoms

RDC diagnosis	*No.*	*Narrow*[1]	*Wide definition only*	*% by either definition*
Schizophrenia	15	11	0	73.3
Schizoaffective manic	1	0	0	0
Schizoaffective depressed	5	2	1	60
Manic disorder	9	0	0	0
Hypomanic disorder	3	0	0	0
Major depressive disorder	29	0	2	6.8
Minor depressive disorder	17	0	0	0
Other	20	0	0	0

1. Includes subjects with both narrow and wide symptoms.

TABLE II

Carpenter score, New Haven Index diagnosis and first-rank symptoms

	Narrow[1]	*Wide only*
Carpenter score of 6 or more (94% probability of being schizophrenic)	7	1
Carpenter score of 5 (78% probability of being schizophrenic)	4	0
Carpenter score of 4 or less (under 62% probability of being schizophrenic)	2	2
New Haven Index positive for schizophrenia	12	2
New Haven Index negative for schizophrenia	1	1

1. Includes subjects with both wide and narrow symptoms.

TABLE III

The relation between three definitions of schizophrenia and presence of narrow and wide first-rank symptoms

No. of definitions of schizophrenia fulfilled	*Narrow (n = 13)*	*Wide only (n = 3)*
Three definitions of schizophrenia	9	0
Two definitions of schizophrenia	4	1
One definition of schizophrenia	0	1
No definition of schizophrenia	0	1

Subjects are ranked according to the number of definitions they fulfil. A score of 5 or more on the Carpenter scheme is deemed to be a diagnosis of schizophrenia.

1. Includes subjects with both wide and narrow first-rank symptoms.

symptoms had an RDC diagnosis of schizophrenia; two subjects with major depressive disorder had first-rank symptoms. Three subjects with first-rank symptoms fulfilled criteria for schizoaffective disorder. No subject with a non-psychotic disorder had a first-rank symptom.

Carpenter's system for the diagnosis of schizophrenia uses a numerical score to predict the probability, expressed as a percentage, that a subject has a schizophrenic illness. It makes use of audible thoughts and broadcast thoughts but does not otherwise require the presence of first-rank symptoms to diagnose schizophrenia. The New Haven Index uses a numerical system to diagnose schizophrenia but has only one cut-off point for diagnosis. In Table II the Carpenter score and New Haven Index is shown for subjects with narrow or wide first-rank symptoms. Four subjects had a score of 4 or less on the Carpenter scheme, which means that these subjects have a high probability of not being schizophrenic. Two of these had narrow (both RDC schizophrenia) and two wide definition first rank symptoms (both RDC major depression). Two were New Haven Index negative, one with narrow and one with wide first-rank symptoms.

If we consider first-rank symptoms in diagnoses other than schizophrenia, a significant proportion of subjects with major depressive disorder or schizoaffective disorder, depressed type, had first-rank symptoms (Table I). Subjects who fulfil the criteria for schizoaffective disorder, depressed type, fulfil criteria for major depressive disorder in addition to having significant schizophrenic symptoms. Combining the schizoaffective, depressed type, subjects with those with major depressive disorder, 15% of subjects with a research diagnosable depressive disorder had first-rank symptoms.

No subject with schizoaffective disorder, manic type, hypomania, or mania had first-rank symptoms, a finding at variance with previous literature.

Three criteria for the diagnosis of schizophrenia were employed in this study (RDC, the Carpenter scheme, and the New Haven Index). A score of 5 or more on the Carpenter scheme (a high probability of being schizophrenic) and a score of 4 or above on the New Haven Index are deemed positive diagnoses of schizophrenia. Table III shows the number of subjects with either narrow or wide first-rank symptoms fulfilling one or more of these criteria. Of the 13 subjects with at least one narrow first-rank symptom, nine fulfilled all three definitions. The other four fulfilled two of the three definitions; two of these were RDC negative for schizophrenia but did fulfil criteria for schizoaffective disorder. Of the three subjects with wide first-rank symptoms only, none fulfilled all three definitions of schizophrenia and none fulfilled RDC criteria for schizophrenia. One fulfilled none of the three definitions.

The two subjects with RDC major depressive disorder had wide first-rank symptoms only. Both were Carpenter negative but one was New Haven positive. There is some evidence here that eliminating those with wide first-rank symptoms would make first-rank symptoms more specific to schizophrenia.

First-rank symptoms and organic brain disease

Of the four subjects with organic brain disease excluded from the main sample, one woman with a recent cerebrovascular accident and acute confusion heard voices outside her head talking to each other about her and commenting on her movements and actions. Voice commentary was a fleeting symptom but 'voices in discussion' was present for ten days.

Discussion

The specificity of first-rank symptoms for schizophrenia has been challenged by many studies, notably those of Carpenter *et al* (1973*b*; Carpenter & Strauss, 1974) and Taylor & Abrams (1973). These studies showed that first-rank symptoms were to be found in affective disorder and, in Carpenter's studies, patients with personality disorder and neurosis. Sixteen per cent of this random sample of admissions to a psychiatric hospital had first-rank symptoms. All first-rank symptoms were represented except for delusional perception, which was uncommon in the study by Mellor (1970), suggesting that it is rare. The six most common symptoms in this study were the same as in Mellor's study. Wide-definition first-rank symptoms in isolation did occur but were also found in conjunction with the narrow definition. The median number of symptoms was two, suggesting that these symptoms tend to cluster together.

Of those receiving an RDC diagnosis of schizophrenia, 73% had first-rank symptoms, confirming that they occur frequently enough to be useful in diagnosing this disorder. As in other studies, schizophrenia was diagnosed in the absence of these symptoms. However, five out of those with first-rank symptoms did not have an RDC diagnosis of schizophrenia. Two of those with first-rank symptoms were negative for schizophrenia using the New Haven Index and four scored below five on the Carpenter system. This poor concordance of Schneiderian first-rank symptoms with three other systems for the diagnosis of schizophrenia accords with the findings of Brockington & Leff (1978).

None of the subjects with first-rank symptoms discordant for schizophrenia received a diagnosis of personality disorder or neurosis using the RDC; instead they received a diagnosis of either schizoaffective disorder (depressed) or major depressive disorder, representing 14% (5/34) of the subjects with such diagnoses. One subject fulfilled no definition of schizophrenia, and here there is a definite occurrence of first-rank symptoms in affective disorder; those with a schizoaffective disorder could have a schizophrenic or affective disorder: first-rank symptoms could not be used to determine which disorder is present unless one made a prior assumption that they are specific to schizophrenia. Schneider's contention that these symptoms, when present in a patient without organic illness, are characteristic of schizophrenia is not supported.

If we turn to the division of these symptoms into narrow and wide definition first-rank symptoms, a different picture emerges. Of 13 subjects with narrow-definition first-rank symptoms, nine fulfilled all three definitions of schizophrenia. The other four had either RDC schizophrenia or schizoaffective disorder as well as fulfilling one other definition of schizophrenia. Of the three subjects with wide-definition first-rank symptoms, only two were diagnosed with RDC major depressive disorder, of whom one fulfilled no definition of schizophrenia and the other only one. If we treat as schizophrenic those diagnosed RDC schizophrenia or schizoaffective in addition to meeting at least one other definition of schizophrenia, then all those with narrow-definition first-rank symptoms would meet this criterion. Two with wide-definition first-rank symptoms only would not meet the criterion.

The finding that no subject with schizoaffective disorder, manic type, mania, or hypomania had first-rank symptoms is not in accord with previous studies (Taylor & Abrams, 1973; Brockington *et al*, 1980*b*).

The evidence presented here suggests that making use of narrow rather than wide definition of first-rank symptoms would make these symptoms more specific to schizophrenia, and this would lend considerable support to Schneider's claims. Mellor (1970) used narrow definitions in his sample of psychotic patients, and in an eight-year follow-up of his original subjects, Mellor *et al* (1981) reported 88% stability of schizophrenic diagnosis; this supports the report here that narrow-definition first-rank symptoms are specific to schizophrenia.

Schizoaffectives are best divided into schizodepressives and schizomanics, because they appear to differ in outcome and family history. Schizomanics have been shown to be very similar in outcome and family history to manic patients (Brockington *et al*, 1980*b*). Schizodepressives do not have a uniform outcome and have both schizophrenia and affective disorder in first-degree relatives (Brockington *et al*, 1980*a*). They are, therefore, a heterogeneous group. The demonstration here that different levels of stringency in defining critical psychopathology, i.e. first-rank symptoms, might help in deciding which disorder is present would, if replicated, help considerably in both clinical practice and research. The importance of characteristic psychopathology, rather than other factors such as outcome, in deciding diagnosis has been strengthened by the demonstration by Cloninger *et al* (1985) of a point of rarity between schizophrenia and other disorders using just four symptoms.

However, Kendell & Gourlay (1970) and Kendell & Brockington (1981) have failed to find such a point of rarity between schizophrenia and affective disorder in a mixed psychotic sample using symptoms or outcome. Their findings combined with two other observations on the genetics of schizophrenia have led Crow (1986) to revive the notion that affective disorder and schizophrenia exist on a continuum. One could visualise schizoaffectives as lying on a continuum between typical schizophrenia and typical affective disorder. Koehler (1979) has demonstrated that first-rank symptoms are best seen as a continuum between narrow and wide definitions. That report together with the findings here that narrow first-rank symptoms are more specific to schizophrenia than wide-definition first-rank symptoms, raises the possibility that there is a continuum of psychopathology in parallel with the presumed continuum of psychoses. Stringently defined first-rank symptoms would be associated with the schizophrenia pole and wide-definition first-rank symptoms with the affective pole. At this time it is not possible to decide whether these psychoses are best described as separate entities with a point of rarity or as a continuum. Future studies should attempt to create a bimodal distribution of psychoses using narrow-definition first-rank symptoms and link this with studies of treatment response, family history, and outcome.

One patient with an organic condition had a definite first-rank symptom, a finding reported previously (Cummings, 1985). Trimble (1987) summarised the findings from numerous studies on the incidence of first-rank symptoms in the psychoses associated with temporal lobe epilepsy, and concluded that Schneider's symptoms might be associated with disturbance in the temporal–limbic system. It may be that any disorder, whether functional or organic, affecting this area of the brain would give rise to these symptoms.

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5-HIAA in Cerebrospinal Fluid and Deficit Schizophrenic Characteristics

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Higher CSF 5-HIAA concentrations and lower CSF HVA concentrations have been associated with various measures of slowed motor behaviour and communication in schizophrenic patients. To derive a single, reliable measure of deficit characteristics in schizophrenic patients, we entered four items of the BPRS reflecting negative symptoms, a work history measure derived from the Strauss-Carpenter scale, and three subscale scores of the WAIS-R into a principal-components analysis to derive a single factor score. The CSF 5-HIAA concentrations were within the normal range of values, and correlated directly with this factor score, but CSF HVA concentrations were not associated with the deficit factor score. These findings add support to the hypothesis that brain serotonin function is associated with deficit schizophrenic characteristics.

Schizophrenia is a complex disorder comprised of multiple symptoms and signs. In the last several years, particular interest has been shown in the division of schizophrenic symptoms as positive or negative (Angrist *et al*, 1980; Crow, 1980; Andreasen & Olsen, 1982; Andreasen *et al*, 1986). Negative symptoms are highly correlated with one another, suggesting a cohesive dimension of psychopathology, in contrast to positive symptoms, which intercorrelate less well (Andreasen & Olsen, 1982). Negative symptoms have been associated with other important schizophrenic characteristics such as poor pre-morbid adjustment, poor response to neuroleptics, chronicity, cognitive deficit, structural changes in the brain, and possible developmental abnormalities (Andreasen & Olsen, 1982; Andreasen *et al*, 1986; Crow, 1980; Opler *et al*, 1984). Carpenter *et al* (1985, 1988) applied the term 'deficit' to such symptoms and signs when they are persistent. The cohesiveness and temporal stability of such deficit characteristics might contribute to determining their underlying biological substrates.

In the last two decades, much work has been done to find cerebrospinal fluid (CSF) neurochemical correlates of schizophrenic symptom clusters. With regard to dopamine, Bowers (1974) has presented evidence that CSF concentrations of homovanillic acid (HVA) are inversely associated with the individual negative symptoms of withdrawal and apathy. Similarly, Lindstrom (1985) demonstrated an inverse correlation between CSF HVA concentrations and lassitude and slowness of movement. 5-hydroxytryptamine (serotonin, 5-HT) has also been hypothesised to be related to the pathogenesis of schizophrenia (for review see van Kammen & Gelernter, 1987), and CSF 5-HIAA levels have been shown to be lower in acutely ill patients (Bowers *et al*, 1969), and in patients with more severe positive symptoms (Post *et al*, 1975; Lindstrom, 1985), or motor agitation (Kirstein *et al*, 1976). Gattaz *et al* (1982) reported positive correlations between CSF 5-HIAA concentration and hallucinations, grandiosity, and tension.

Our group could not demonstrate group differences among schizophrenics, patients with major depressive disorder, and community volunteers for CSF concentrations of HVA, 5-HIAA, and 3-methoxy-4-hydroxy-phenylglycol (MHPG) (Berger *et al*, 1980). Since then, we have concentrated our efforts on finding correlations of CSF neurochemical variables with particular dimensions of psychopathology. Here we report our efforts to demonstrate a correlation between CSF HVA and 5-HIAA concentrations and deficit characteristics in schizophrenic patients. We hypothesised that CSF HVA would correlate inversely and CSF 5-HIAA directly with such characteristics. To measure deficit characteristics in the most reliable manner possible, we constructed a single deficit factor score, derived from a principal-components analysis (PCA) of items chosen *a priori* from our database including symptom, work history, and cognitive measures.

Method

Sample selection

The total sample consisted of 36 male in-patients at the VA-Stanford Clinical Research Center at the Palo Alto Veterans Administration Medical Center. Written informed consent was obtained for all procedures. Thirty patients met Research Diagnostic Criteria (RDC) for schizophrenia and six for schizoaffective disorder (mainly schizophrenic subtype) (Spitzer *et al*, 1978). All patients were free of all oral psychotherapeutic medications for at least 14 days at the time of symptom assessment and lumbar puncture, except for occasional doses of chloral hydrate (500–1000 mg). Not all patients were drug-free at the time of cognitive assessment using the WAIS–R (see below). No patient had been given the depot form of any neuroleptic for at least four months before their participation in this study.

Psychopathology, demographics, and cognitive assessment

All patients were evaluated using the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1963). Four BPRS items were selected as measures of negative symptoms: emotional withdrawal, blunted effect, motor retardation, and mannerisms and posturing. The justification for selecting these items was based upon clinical opinion and published BPRS factor analyses; the first three are classically grouped into a BPRS retardation-withdrawal factor (Overall, 1974). Although 'mannerisms and posturing' are not usually classified as a negative symptom, we felt they may represent an alternative manifestation of autistic withdrawal in schizophrenia, and therefore should be included among the negative symptoms. Further, they intercorrelate with the BPRS negative symptom items in several published factor analyses (Guelfi *et al*, 1989). In prior work, we found a global assessment of negative symptoms, using the BPRS, to be comparable with global measures of negative symptoms from the Scale for the Assessment of Negative Symptoms developed by Andreason (Thiemann *et al*, 1987).

In 35 of the 36 patients, the ratings were performed independently by a specially-trained research assistant and psychiatrist, or by two specially trained research assistants following a single joint interview. In those cases, all scores represent the mean of the two raters. In one case, the rating was performed by a single interviewer. Inter-rater reliability for the BPRS is assessed in an ongoing fashion at our centre, and for the sum of these four symptom items the value is $r_s = 0.71$ (Guelfi *et al*, 1989).

Historical data needed to score the work history item of the Strauss-Carpenter scale (Strauss & Carpenter, 1974) were collected from each patient at admission by a specially trained research assistant. This item was chosen for inclusion in the analysis because: (a) it added a longitudinal dimension to our assessment of deficit characteristics; (b) Strauss & Carpenter (1974) found it to be among the most informative items in their prognosis scale; and (c) the data needed to score this item could be reliably obtained in our patients.

The WAIS–R was also administered to each patient, by one of the authors (JAM). The WAIS–R is a standardised measure of cognitive performance that has received extensive psychometric validation (Lezak, 1983). Three subtests were chosen from the 11 WAIS–R scales as potential measures of deficit characteristics: digit symbol, digit span, and picture arrangement. Digit symbol is sensitive to deficits in psychomotor speed, sustained attention, and visuomotor co-ordination (Lezak, 1983), and has been linked to a poor outcome and negative symptoms in schizophrenia (Rubin *et al*, 1987, unpublished conference presentation). Digit span is a task requiring attention span and concentrated effort (Matarazzo, 1972). Finally, picture arrangement requires skills in comprehending a situation, thinking in a sequential manner, and being aware of social behaviour and relationships (Lezak, 1983). Prior factor analytic work (Parker, 1983; Atkinson & Cyr, 1984) has noted that these scales are components of the freedom-from-distractibility factor extracted in three-factor solutions of the WAIS–R. We reasoned *a priori* that performance on all three of these WAIS–R subtests would be decreased in patients with other deficit characteristics.

Not every patient was free of oral psychotherapeutic medications for at least 14 days at the time of WAIS–R administration; only 19 patients met this criterion; three were 1–14 days drug-free, and 14 were being prescribed neuroleptic medication. Antipsychotic medications have been found to have equivocal effects on WAIS–R performance (Heaton & Crowley, 1981); however, we felt that inclusion of WAIS–R data both drug-free and during neuroleptic treatment introduced no bias in favour of our hypothesis.

Lumbar puncture and CSF analysis

Twenty-one patients had a lumbar puncture. All lumbar punctures were also performed at least 14 days after the patient was free of medication, except for occasional doses of chloral hydrate in some patients up to 12 hours before the lumbar puncture. The lumbar puncture was performed at approximately 8 a.m. after an overnight fast, in the lateral decubitus position. In a few cases, when a successful lumbar puncture could not be performed in the lateral decubitus position, the patient was briefly raised to a sitting position. Evidence suggests that monoamine metabolite concentrations are less influenced by the position of the patient during the tap than by the CSF aliquot chosen for analysis (Gateless *et al*, 1984). The fraction of CSF corresponding to the 2nd to 6th ml was collected in a plastic tube containing 4 μmol of ascorbic acid and stored at −80°C until assay.

Nine male community volunteers who responded to a newspaper advertisement also underwent a lumbar puncture to obtain CSF comparison data. They were screened by a specially trained research assistant using the Schedule for Affective Disorders and Schizophrenia (lifetime version) (SADS–L; Endicott & Spitzer, 1978) to rule out any psychiatric diagnosis defined by the RDC, and interviewed and examined by a physician to rule out any medical or neurological disorders that could potentially confound neurochemical measurements. Their CSF 5-HIAA and HVA concentrations are included in this report for reference only, as the principal hypothesis was not based upon group comparisons.

The CSF concentrations of 5-HIAA and HVA were determined by selected ion monitoring using gas chromatography/mass spectrometry (Faull *et al*, 1984). The samples were analysed in batches within three months of collection.

TABLE I
Description of patient populations (means ±s.d.)

	Total sample (n=*36*)	*Lumbar puncture* (n=*21*)	*Volunteer* (n=*9*)
Age: years	35.8±9.0	37.6±10.9	33.0±15.8
Age at onset: years	23.5±6.0	23.2±7.0	–
Duration of illness: years	12.8±9.5	14.3±9.7	–
BPRS			
emotional withdrawal	2.8±1.2	2.7±1.1	–
blunted affect	2.8±1.2	2.8±1.1	–
mannerisms/posturing	1.2±0.4	1.2±0.4	–
motor retardation	1.7 - 1.0	1.6±0.8	–
Work history	1.3±1.6	1.0±1.4	–
WAIS			
digit symbol	5.8±1.6	5.7±1.6	–
digit span	8.1±2.8	8.3±3.0	–
picture arrangement	7.8±3.4	7.6±3.2	–
CSF 5-HIAA: nmol/dm^3	–	90.0±34.3	84.8±34.7
CSF HVA: nmol/dm^3	–	167.9±55.8	174.7±76.2

Calculation of the quantitites of 5-HIAA and HVA was made by interpolation from standard calibration curves constructed from data obtained from concurrent standard samples. Because the data were collected over 1983–86, precautions were taken to ensure that batch effects were minimised and there was no trend in the quantitative measurements with time. The standard curves were prepared using the same sample of authentic 5-HIAA, which was stored in crystalline form at −4°C, checked periodically using high-pressure liquid chromatography coupled to electrochemical detection.

Data analysis

Principal-components analysis (Maxwell, 1977) was used to perform data reduction on the selected variables, all of which were hypothesised to reflect deficit characteristics in schizophrenia. To include the largest number of patients in this factor determination, all patients with BPRS ratings, the Strauss-Carpenter item, and WAIS–R subscale scores ($n=36$) in our database were analysed. It should be borne in mind that this number of subjects would not permit us to state a definite factor structure of these variables (see Results). In the lumbar puncture sample, factor 1 scores were correlated (Spearman) with CSF 5-HIAA and HVA values to test the primary hypotheses. Individual Spearman rank-order estimates of the correlations between the BPRS measures, the work history item, and the WAIS–R subscales with CSF 5-HIAA were also performed. Spearman rather than Pearson estimates were made because we wished to minimise our reliance upon the assumption of a normal distribution for any of the variables entered into correlation estimates. Only the principal-components analysis necessitated this assumption; it was performed for data reduction only, and not for testing the hypothesis. Since we hypothesised no group differences, comparisons between CSF 5-HIAA and HVA values for the patient and community volunteer groups were made using 95% confidence intervals on the differences between the means.

Results

A description of the total sample, the lumbar puncture sample, and the volunteer sample is given in Table I. Mean CSF 5-HIAA and HVA concentrations for the community volunteers compared with the patients were similar (for 5-HIAA, difference is 5.2±48.8 nmol/dm^3; for HVA, difference is 6.8±94.4 nmol/dm^3). The CSF comparison group was also similar to the patients with respect to age.

The outcome of the principal-components analysis revealed that a single factor, designated factor 1 (eigenvalue 2.9), carried 36% of the variance. Individual component loadings for factor 1 are shown in Table II. Second (eigenvalue 1.5) and third (eigenvalue 1.2) factors were also found, but these were not entered into further analyses because of the lower eigenvalues obtained. Table III illustrates estimates of the inter-correlations among the items that comprised factor 1.

TABLE II
Individual component loadings for factor 1

BPRS	
emotional withdrawal	0.82
blunted affect	0.85
mannerisms/posturing	0.53
motor retardation	0.53
Work history[1]	−0.41
WAIS	
digit symbol[1]	−0.48
digit span[1]	−0.55
picture arrangement[1]	−0.47

1. Decreases in these scores were correlated with increasing CSF 5-HIAA concentrations

TABLE III
Inter-relationships (Spearman's r) among items comprising factor 1

		WAIS		*Useful work*		*BPRS*	
	Picture arrangement	*Digit span*	*Digit symbol*		*Mannerism/ posturing*	*Motor retardation*	*Blunted affect*
BPRS							
emotional withdrawal	0.16	0.28	0.30	0.24	0.34	0.43	0.79
blunted affect	0.20	0.33	0.24	0.23	0.22	0.56	–
motor retardation	−0.07	0.07	0.05	0.07	−0.04	–	
mannerisms/posturing	0.20	0.11	0.38	0.36	–		
Useful work	0.43	−0 02	0.19	–			
WAIS							
digit symbol	0.38	0.51	–				
digit span	0.22	–					
picture arrangement	–						

The direction of scores for useful work, digit symbol, digit span, and picture arrangement were reversed for these calculations, so that the applicable correlations appear as positive.

The preponderance of these were positive, adding further support to the *a priori* choice of these items, and the need for a data reduction procedure (i.e. principal-components analysis).

The significant correlation between factor 1 scores for each patient with a lumbar puncture ($n = 21$) and CSF 5-HIAA values is illustrated in Fig. 1. The correlation between factor 1 scores and CSF HVA values ($r_s = -0.11$) was not significant. Table IV summarises the individual correlation estimates of CSF 5-HIAA with each of the deficit symptom measures derived from the BPRS, the Strauss-Carpenter work history variable, and the WAIS-R variables. (For reference, for one-tail significance $P < 0.05$, r_s must be > 0.38.) Relationships were apparent between many of the individual measures entered into the principal-components analysis and CSF 5-HIAA concentrations, although none were as strong as with the factor 1 score. The individual correlations may have been more attenuated by the unreliabilities of the individual measures compared with the reduced factor 1 score.

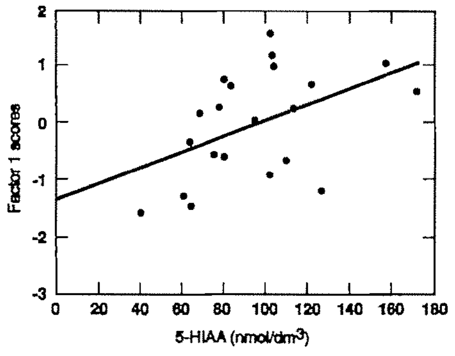

FIG. 1 Scatterplot of CSF 5-HIAA versus factor 1 scores ($r_s = 0.50$, $P < 0.05$).

TABLE IV
Correlations (Spearman's r) of CSF 5-HIAA with individual deficit variables (n = 21)

Deficit variable	r
BPRS	
emotional withdrawal	0.26
blunted affect	0.31
mannerisms/posturing	0.16
motor retardation	0.11
Work history[1]	0.41
WAIS	
digit symbol[1]	0.41
digit span[1]	0.44
picture arrangement[1]	0.25

[1] Decreases in these scores were correlated with increasing CSF 5-HIAA concentrations.

Discussion

The relationship between CSF 5-HIAA and factor 1 scores found here is consistent with previous findings. Kirstein *et al* (1976) found that there was an inverse correlation between the accumulated concentrations of CSF 5-HIAA after probenecid administration and psychomotor agitation in ten schizophrenic patients. Post *et al* (1975) found that CSF 5-HIAA accumulations after probenecid were also correlated with hallucinations and delusions, described as Schneiderian in quality, in 20 patients. Bowers (1978) has also demonstrated an inverse correlation between CSF 5-HIAA and favourable prognostic signs, acute course, and low levels of emotional withdrawal. Lindstrom (1985) found that CSF 5-HIAA concentrations (without probenecid) were inversely correlated with delusions. Previous preliminary work by our group (King *et al*, 1985), in a non-overlapping group of 21 schizophrenic patients, found that the log of CSF 5-HIAA concentrations was directly correlated with mannerisms and posturing (rated by the BPRS), a symptom in our population that covaries with other negative symptoms (Guelfi *et al*, 1989). In sum, the literature and these data suggest that lower concentrations of CSF 5-HIAA may be associated with psychomotor agitation and positive schizophrenic symptoms, while higher CSF 5-HIAA concentrations are associated with negative symptoms and chronicity within groups of schizophrenic patients.

The CSF 5-HIAA and HVA concentrations were themselves correlated ($r_s = 0.53$, $P < 0.02$) in this study. This inter-relationship has been observed previously in other samples of community volunteers, patients with major depressive disorder, as well as schizophrenics (Faull *et al*, 1984), although no satisfactory explanation for it has been found. It is important to note that despite the correlation between these CSF variables, only CSF 5-HIAA correlated with factor 1 scores. In fact, when a partial correlation coefficient was estimated between CSF 5-HIAA concentrations and factor 1 scores, controlling for CSF HVA, it remained equivalent ($r_s = 0.52$, $P < 0.05$).

The relationship between CSF 5-HIAA concentrations and deficit characteristics observed here was found with the 'normal' range of CSF 5-HIAA values. In general, group comparisons between schizophrenics and controls that are either normal or diagnosed with a mood disorder have been negative (Bowers, 1974; Post *et al*, 1975; Kirstein *et al*, 1976; Berger *et al*, 1980; Lindstrom, 1985). Only two studies have shown comparatively low CSF 5-HIAA concentrations in schizophrenics (Bowers *et al*, 1969; Gattaz *et al*, 1982), while one has shown high CSF 5-HIAA concentrations (Gerner *et al*, 1984). The differences in the range of 5-HIAA values observed among schizophrenics in these studies might have been due to a relative preponderance of positive or negative symptoms.

Associations between CSF 5-HIAA and other features of schizophrenia may exist. Higher CSF 5-HIAA concentrations have been associated with a family history of schizophrenia (Sedvall & Wode-Helgodt, 1980). Lower CSF 5-HIAA concentrations have been associated with suicidal behaviour in schizophrenics (van Praag, 1983; Ninan *et al*, 1984) and with lateral ventricular enlargement (Potkin *et al*, 1983; van Kammen *et al*, 1985), or cortical atrophy (van Kammen *et al*, 1985). These latter findings may be at odds with the finding presented here. It seems reasonable to expect a correlation between deficit characteristics and cerebral atrophy in schizophrenia, as hypothesised by Crow (1980) in his type I/type II model of the disease. Some (Andreasen & Olsen, 1982) but not all investigators (Nasrallah *et al*, 1983; Pfefferbaum *et al*, 1988) have detected a correlation between signs of cerebral atrophy and deficit characteristics. An interesting facet of the problem is to consider the effect a larger total CSF volume would have on CSF 5-HIAA concentrations, given no change in the absolute quantity of CSF 5-HIAA. We are currently undertaking a study to address this issue.

The present work does not support prior findings of relating deficit characteristics in schizophrenics with lower CSF HVA concentrations (Bower, 1974; Lindstrom, 1985), although we did not use the same clinical instruments. Also, our patients had been treated for many years with neuroleptics and had been recently withdrawn from them. Such treatment may have added another source of variance to our CSF HVA measurements.

There is no established method for combining measures, such as those used to assess deficit characteristics in this study, into a single variable. We wanted to avoid testing the statistical significance of a long list of bivariate correlations between CSF 5-HIAA and HVA concentrations and the predictor variables, many of which were inter-related. This approach would have necessitated large corrections in the level of significance for *P* values. Co-linearity among the variables would confound the use of multiple-regression procedures. After careful consideration, we chose to employ principal-components analysis to reduce the many measures of deficit characteristics into a single score. However, our use of a single factor score should not be construed as a test of cohesiveness among deficit characteristics. The work of other investigators has better addressed this issue (Andreasen & Olsen, 1982; Carpenter *et al*, 1988). In fact, the small number of subjects available precludes conclusions about the component loadings shown in Table II. As an alternative, we also transformed each measure for each patient into a *Z* score, and averaged all *Z* scores for each patient to yield a mean *Z* score reflecting the deficit characteristics. The correlation between these mean deficit *Z* scores and CSF 5-HIAA concentrations was equivalent, $r_s = 0.54$, suggesting that the relationship between the factor 1 scores and CSF 5-HIAA was not dependent upon the loadings given to each measure by the principal components analysis.

Conclusions

The findings in this report add support to the hypothesis that 5-HT plays an important role in schizophrenia. In particular, the data suggest that 5-HT function within the normal range may be linked to deficit characteristics in schizophrenic patients. Cause and effect issues remain unclear. Further, these findings support recent preliminary reports that pharmacological agents that block or deplete 5-HT may improve such symptoms (Ceulemans *et al*, 1985; Stahl *et al*, 1985). A reasonable next step may be to determine whether decreases in deficit symptoms during such treatment correlate with decreases in CSF 5-HIAA concentrations.

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Life Events and the Onset of Mania

PAUL SCLARE and FRANCIS CREED

Thirty manic in-patients were interviewed in hospital using the LEDS, and 24 were re-interviewed 6–12 months after discharge. Data for life events were analysed by: comparing events before onset of mania and before re-interview; and comparing these manic patients with patients in other studies which examined life events and the onset of schizophrenia and depression. No relationship was found between life events and the onset of mania in this preliminary study. Previous studies reporting a link between events and the onset of mania have serious methodological flaws, and definitive data have yet to be produced.

There is a well established link between life events and the onset of depression (Brown & Harris, 1978) and of schizophrenia (Brown & Birley, 1968; Jacobs & Myers, 1976; Day *et al*, 1987), but the results concerning mania are less clear, largely because of the methodological problems involved. Although Bleuler (1951) and Schneider (1959) both categorically stated that mania is never reactive, anecdotal case studies have appeared to support a link between the onset of mania and a number of specific life events, such as bereavement, natural disasters, and road traffic accidents (Rickarby, 1977; Hollender & Goldin, 1978; Sinanan, 1984; Aronson & Shukla, 1987). Of seven recent studies designed specifically to examine the relationship between life events and the onset of mania, most appear to have produced a positive result (Leff *et al*, 1976; Hall *et al*, 1977; Patrick *et al*, 1978; Ambelas, 1979, 1987; Dunner *et al*, 1979; Kennedy *et al*, 1983).

However, two major methodological problems have not been addressed in most of the studies to date. Firstly, there has been little attempt to date the onset of mania accurately. In three of the seven recent studies the authors assumed that onset of mania coincided with admission to hospital. In order to justify this assumption, one author stated "the delay between onset [of mania] and admission was usually very brief" (Ambelas, 1987). Secondly, there have been large differences in the methods used to collect life event data. Three studies relied on information gleaned from clinical case notes. A further three studies used a check-list to collect information on life events, with patients expected to recall events occurring many years before interview. Only one study accurately dated the onset of mania and used the most rigorous instrument available – the Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978) – to collect life event data. This study failed to find a relationship between life events and the onset of mania (Chung *et al*, 1986).

The present study used the LEDS to establish life events during the six months before the onset of mania and for six months after recovery when the patient was clinically well. In this way each patient acted as his/her own control. This is appropriate as there is no obvious control group of people whose lives have been disrupted by a similar illness. The results are also compared with published studies which have used the LEDS in depression and in schizophrenia (Brown & Birley 1968; Brown & Harris, 1978).

Method

Subjects

A consecutive series of patients with mania was collected by screening all acute admissions to three district general hospital psychiatric units in Manchester between February 1986 and September 1987. For inclusion, patients had to have a clinical diagnosis of mania and to have recovered from any previous episode of either depression or mania at least nine months before admission for the present manic illness. Patients with a possible organic aetiology, with a possibility of schizo-affective disorder, or with evident organic impairment of memory, were excluded from the study. Thirty-four patients fulfilled the inclusion criteria, of whom four declined to take part in the study. It was possible to re-interview 24 of the 30 subjects six months after recovery.

Assessments

Patients were interviewed on two occasions. The first interview took place during the hospital admission, but only after the patient's mental state had returned to normal. The second interview was carried out in the patient's home, 6–12 months after discharge.

Psychiatric symptoms were assessed by administering the Present State Examination (PSE; Wing *et al*, 1974). At the first interview this allowed us to confirm the clinical diagnosis of mania and to determine its onset with accuracy. The onset of each manic symptom was dated, and the earliest of these was taken as the date of onset of the illness. At the second interview, the PSE questions were applied to the previous six months to confirm that the patient had remained clinically well since discharge.

Life events data were collected using LEDS, a semi-structured interview which elicits important events and rates their severity (Brown, 1974; Brown & Harris, 1978; Neilson *et al*, 1987). Great care was taken in this study to date the life events with accuracy, and they were rated for degree of threat by experienced raters blind as to whether the results came from the first or second patient interview. The independence of events was rated, and any illness-related events were excluded from the analyses. The LEDS was administered at both interviews, and patients' accounts of life events (and psychiatric symptoms) were confirmed with patients' relatives whenever possible. The case notes were scrutinised after the first interview to check whether the date of onset of symptoms elicited from the patient/relative coincided with that recorded in the notes.

Results are presented in terms of the number of subjects experiencing an event over each time period, firstly comparing the six months before onset with the six months before the follow-up interview, and secondly with the results of other studies using the LEDS to examine depression and schizophrenia (Brown & Birley, 1968; Brown & Harris, 1978).

Statistical analysis used the χ^2 test, using the McNemar test to examine for significance of change between interviews.

Results

Of the 30 patients recruited (16 women, 14 men, aged 18–70 years, mean 41 years), 24 completed both interviews. Of the remainder, one was unavailable, two became ill again, two refused, and one had died. As a result of the first PSE five were rejected: three had mixed affective states, and two had schizophrenia.

Onset of mania

The mean age when subjects experienced their first episode of mania or depression was 29 years (range 13–53 years), and they had had 0–23 (median 4) previously documented episodes of affective illness (mania or depression) requiring hospital admission. The time between onset of the current episode of mania and the first interview was 18–158 days (median 54 days). Relatives of 15 of the 30 patients were interviewed, and it was found that the patients' own reports of both onset and events were accurate. The interviews produced a date of onset earlier than that recorded in the notes for five patients, although no clear date of onset was recorded in many of the notes. The symptoms of elation, overactivity, decreased sleep, overspending, and sexual indiscretions were reported more frequently by the patient than had been recorded in the notes. The reverse was true for irritability, racing thoughts/pressure of speech, and flight of ideas.

For the present illness, the time between the appearance of the first symptoms of mania and admission varied from two days to over four months (median 22 days). The first symptoms of illness found to be common at onset are listed in Table I. In 13 cases the onset was marked by three or more symptoms that occurred simultaneously, in 12 cases there were two symptoms at onset, and in five cases the onset was marked by a single symptom.

TABLE I
Symptoms in the onset of mania

	No. of patients experiencing symptom (elicited by PSE)	*No. of times occurred as first symptom*
Increased activity	24	16
Elated mood	23	15
Reduced sleep	22	15
Grandiosity/increased self-esteem	13	6
Irritability	12	8
Excess spending/	11	6
sexual indiscretions	11	6
Racing thoughts	5	3

Of the 17 patients receiving prophylactic treatment with lithium carbonate in the year before onset of illness, two stopped taking lithium shortly before the onset of mania.

Life events

Figure 1 presents data for all 'independent' and 'possibly independent' events in the two six-month periods, for the 24 patients who were interviewed on two occasions. It is clear that there is no consistent difference between the two time periods, and no trend emerges. A similar analysis was done for severe events only. A severe event is one which is rated as 1 or 2 on long-term threat, and in which the subject is the focus. Again, there was no apparent difference in the rate of events in the two periods studied.

Statistical analysis essentially confirms the above findings. The results for different types of events over three time periods (3, 13 and 26 weeks) before onset/second interview are presented in Table II. The only significant difference to emerge concerned the proportion of subjects who had experienced an 'independent' or 'possibly independent'

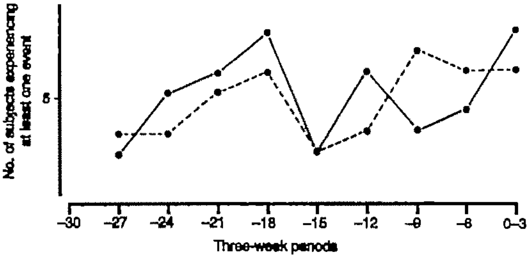

FIG. 1 Number of subjects experiencing any event in each three-week period before onset (—) and before second interview (---) Data presented only for 24 subjects interviewed twice.

TABLE II

No. of subjects experiencing a life event over three time periods (3, 13, 26 weeks) comparing six months before onset and six months before follow-up interview (results for 24 patients interviewed after onset and after discharge).

Period before interview	*Any event*		*Threatening event*			
			Short term		*Long term*	
	1st interview	*2nd interview*	*1st interview*	*2nd interview*	*1st interview*	*2nd interview*
Independent events only						
3 weeks	5	2	3	1	1	0
13 weeks	9	11	3	7	1	4
26 weeks	14	14	8	8	6	5
Independent and possibly independent events						
3 weeks	8	6	4	2	1	0
13 weeks	16	12	7	8	3	4
26 weeks	22	14*	13	9	11	5

*$P = <0.05$.

TABLE III

No. of subjects with at least one life event for each category in 26 weeks before onset for three groups of patients, defined in terms of time between first episode of affective disorder and present admission

	Independent and possibly independent			*Independent events only*
	Any event	*Threatening event*		
		Short term	*Long term*	*any event*
Group A[1] ($n=10$)	9	7	6	4
Group B[2] ($n=9$)	7	6	3	5
Group C[3] ($n=11$)	10	4	3	6
	–	–	–	–
Total ($n=30$)	26	17	12	15

1. First episode within 5 years of present episode.
2. First episode within 5–15 years of present episode.
3. First episode more than 15 years before present episode.

event during the longest time period (26 weeks) before onset compared with a similar period following recovery.

In order to compare our events with those of Ambelas (1987), the life events of recent onset manics, compared with those with many previous episodes, are displayed in Table III. There was a trend for those subjects whose first episode of illness was less than five years previously (group A) to have experienced more threatening events. We compared the duration of disorder with number of confidants, and found a significant negative correlation (Spearman's rho -0.45, $P<0.01$); the older patients with many previous episodes had very small social networks (average of 1 confidant each compared to 2.5 for those with shortest time since first episode). This means they had less chance of experiencing a life event affecting themselves or close confidant.

The pattern of life events was compared for the subjects taking lithium and the remainder. The pattern was practically identical for these two groups.

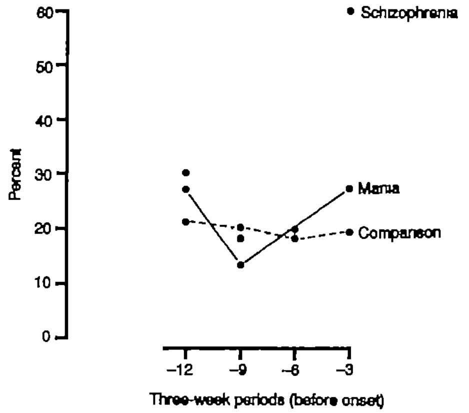

FIG. 2 Number of subjects experiencing a life event (expressed as percentage) during four 3-week periods before onset of mania (present study $n=30$), schizophrenia ($n=50$), or before interview for community comparison subjects ($n=50$) (Brown & Birley, 1968).

Comparison with schizophrenia and depression

Brown & Birley (1968) compared a sample of schizophrenic patients with general population controls. Their results

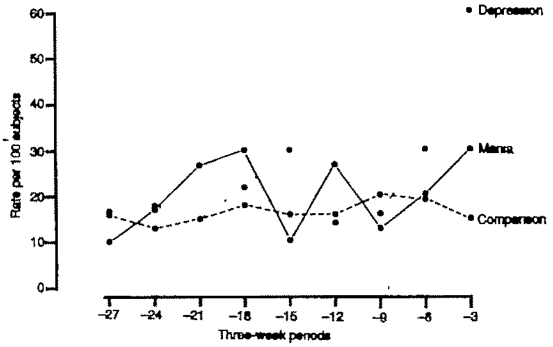

FIG. 3 Number of subjects experiencing a life event (expressed as rate per 100 subjects) in each 3-week period over 6 months before onset of depression or to interview for community comparison subjects (Brown & Harris, 1978) and before onset of mania in present study.

indicated a marked increase in the rate of life events confined to the three weeks before the onset of schizophrenia. Figure 2 compares our samples of 30 manic subjects with the 50 schizophrenic patients and control group described by Brown & Birley. Our manic subjects did not show an excess of life events in the three weeks before onset of illness.

Brown & Harris (1978) reported that depressed women showed an increase in the rate of severely threatening life events in the three weeks before the onset of depression when compared with matched population controls. Figure 3 compares our sample of manic patients with the depressed women and controls reported by Brown and Harris. The manic subjects did not show an increase in the rate of severe events before the onset of illness.

Discussion

The execution of this study was more difficult than we had anticipated, for two reasons. Firstly, it was difficult to find subjects who fulfilled our inclusion criteria. Many patients admitted to hospital for treatment of mania had not been clinically well over the previous nine months, and we could not be sure that they were in a stable state before onset. Other patients had to be excluded because the PSE revealed that they had marked schizophrenic features or a mixed affective state; they were excluded because we wished to compare our results for mania with those for schizophrenia and depression. Secondly, the interval between onset of mania and hospital admission was longer than we had expected. This meant that many life events shortly before admission occurred after the onset of mania and therefore had to be excluded from the study. Two examples will illustrate the problem.

One example was the arrest of a man who stole a wreath from a public memorial. He did this after his initial symptoms of elation, irritability, and sleep impairment had commenced, but the arrest, court case, and subsequent imprisonment all occurred between onset and admission. Another example was a woman who left her husband, returned to England, and filed for divorce. This sequence of events started one week after her symptoms of increased activity, inflated self-esteem, elation, and impaired sleep had begun, but was completed before she was admitted.

Such examples make it clear why previous studies that have assumed hospital admission to be synonymous with onset have found an excess of life events when we have found none. The median time between onset of first symptom and hospital admission in this study was 22 days, whereas Ambelas (1979, 1987) examined events only during the 28 days before admission (interpersonal conflicts, difficulties at work). It is likely that the events collected by Ambelas were a result of the illness rather than contributing to its cause. The study of Kennedy *et al* (1983) was the only one to show an excess of independent events during four months before admission.

We used the conservative measure of first hypomanic symptom as the point of onset. The median times between onset and admission of 22 days is remarkably similar to that of Winokur *et al* (1969), who recorded a mean time of 29.8 days for 100 episodes of mania. For the patients of the present study who had a longer duration between onset and admission it is possible that there was a marked change in the hypomanic state closer to the time of admission. Our study was not designed to assess this because of the enormous difficulty of deciding whether life events are related to the illness, when they occur *after* the first symptoms have developed. A study which examined such 'change points' would allow one to assess whether events exacerbated a hypomanic state, but such events could not be considered causal if they had occurred after the first symptom.

There are a number of additional reasons why this study must be considered a preliminary methodological one, rather than a definitive one. The first limitation was the small size of the sample. Although the LEDS is the most robust measure of life events, the rate of severe life events is low in the general population (only 17–20% of women experience a severe event over a 26-week period) (Brown & Harris, 1978, Miller *et al*, 1986). So a sample twice the size of ours would be required to produce a definite result. The sample size was limited for the reasons mentioned above, and it may have been that those patients whose illness is most likely to be triggered by life events were excluded. Chung *et al* (1986) found that those with schizophreniform psychosis had an onset more closely related to life events than those with hypomania.

Aranson & Shukla (1987) found that hypomanics who had not been stable over previous weeks or months were those most likely to become ill when faced with a stressful event. These authors were able to study patients attending a lithium clinic at the time of a catastrophic hurricane – the archetypal severe and 'independent' event. There was a significant increase in relapse rate in the subsequent two weeks; 10 of 69 patients relapsed. Half of these ten were hypomanic or manic relapses, the rest were depressed. Of the ten patients who relapsed, seven had previously been in an unstable state – most were not euthymic at the time of the hurricane, and six were still having their neuroleptic medication reduced after being ill. None of these patients would have been included in our study, which required patients to be euthymic for at least six months before the onset of the index illness. In their study only five patients out of 60 became hypomanic or manic after a hurricane. One possible reason for this was the fact that all were on lithium at the time. In the present study just over half of the patients were taking lithium when they became ill; the proportion was almost identical at the follow-up interview. Our study was not designed to examine the effect of lithium – such a study would require patients on lithium exposed to stressful events but without becoming ill.

The only significant increase in severely threatening events was during the time period furthest from the onset of mania (13–26 weeks before onset), whereas any clear link, akin to that established in schizophrenia and depression, would have been expected close to onset. We had not expected this negative result, so we checked our methodology. The interviews (on audiotape) were rechecked by the senior author (FC) to ensure that life events were not being missed, and we examined different types of events. Events with threat in the short term were examined in case they caused enough arousal in a patient vulnerable to develop an episode of mania at that point in time (Aronson & Shukla, 1987), similar to the pattern found in appendicitis (Creed, 1981). The overall rate of events was 164.8 per 100 persons per six months, comparable to the rates of 166, 159.7, and 150 quoted by Miller *et al* (1986). The proportion of all events rated as severe was 20% compared to 16% of Brown & Harris (1978).

We have also been stimulated to carefully re-evaluate previous published studies. It can be seen from Table IV that there has been little consistency in the methods used or the period considered before onset. There is a clear negative correlation between the sophistication of methodology and numbers studied. Only one other study has used the LEDS instrument to measure life events and examined events before onset rather than admission (Chung *et al*, 1986), and those authors also found that 14 manics were less likely than 15 schizophrenics to have experienced a severely threatening event before onset.

Previous studies have used a variety of control groups, surgical controls being the most frequent; only one other study has used the patients as their own controls. Our study did not include a separate control group, partly because of the limited time available for interviews, but we also felt that a before/after design was better as those subjects who had many previous episodes of manic-depression were not easily matched with a comparison group. In fact, such patients had a very restricted social network, which is partly responsible for their low rate of life events. A reduced number of confidants was also observed by Kennedy *et al* (1983), but in that study those wtihout a confidant were more likely than the remainder to have experienced an undesirable event – the reverse of our finding.

The results have been presented in this study as the numbers of subjects who had experienced an event rather than as a total number of events reported by each group. This is the established way to present results using the LEDS, and the reasons for this have been discussed by Brown & Harris (1978, p. 107). Kennedy *et al* (1983) found that similar numbers

TABLE IV

	No. of patients	*Controls*	*Time period (weeks)*	*% with event*	*Methodological problems*
Leff *et al* (1976)	63	None	13	28%	Anecdotal
Ambelas (1979)	67	Surgical	4	28% v. 4%	Case notes study
Ambelas (1987)	50 (first illness)	Surgical	4	66% v. 8%	Case notes study
Kennedy *et al* (1983)	20	Own/surgical	16	85% v. 70% (independent severe 29% v. 33%)	Late interview
Chung *et al* (1986)	14	Schizophrenia/ schizoaffective	26	14% v. 33% v. 66%	Small numbers

of manics before and after admission had experienced an event; it was the rate of severe events that was different.

Would using rates of events, rather than proportion who had experienced an event, made a difference? Caution must be exercised when extrapolating from small numbers to rates per 100 persons per six months, but when this was done for the present data the rates before onset were 190 per 100 persons per six months and 133 per 100 persons for the six months after discharge. These figures exclude events related to the illness, for example children being taken into care, divorce, daughter going to a child psychiatrist, and news of examination failure, all of which were probably the result of the patient's actions when ill. So there was a trend towards more events before onset, although, as mentioned earlier, this excess was not immediately before onset. There were no other differences when comparing events before and after: 34/57 (59%) of events before onset were rated as possibly independent, compared with 16/28 (57%) of those after discharge. Rates of events emphasised the low rates of events for those with many previous manic illnesses. The rates for groups A,B,C in Table III were 225, 130, and 90 per 100 persons per six months respectively (compared to the 'norm' of 160), which suggests that these subgroups of manic patients may best be considered separately in future research.

Aranson & Shukla (1987) also concluded that there were subgroups of manics: a small group (14% of their lithium clinic attenders) who were susceptible to environmental stress, and a larger group (86%) who were stable, remaining well in spite of the catastrophic nature of the hurricane and its consequences. It would be the latter who corresponded to the subjects included in the present study.

The subgroup of the greatest interest in the present project was that including those patients with a relatively recent onset (group A in Table III). Six of these ten patients had experienced a severe life event over the previous six months, which is similar to the proportion of depressed patients (Brown & Harris, 1978), twice the proportion for the remaining 20 subjects of the present study, and considerably more than for the 14 manic patients studied by Chung *et al* (1986), two of whom had experienced a severe life event. However, all but two of these events were rated as possibly independent (leaving home after a row, ending relationship with a girlfriend, leaving husband, ending relationship with boyfriend), which raises the theoretical possibility that prodromal features of the illness could have caused these events rather than vice versa.

This study has led us to consider carefully the problems involved in a study of life events in mania: the size and nature of the sample, the timing of onset, the thoroughness of interviewing, and control groups. These problems must be tackled in a future study if reliable results are to be obtained.

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Familial Tourette's Syndrome in a Large British Pedigree
Associated Psychopathology, Severity, and Potential for Linkage Analysis

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A British pedigree multiply affected by the Gilles de la Tourette syndrome and spanning six generations is described. Of 122 members identified, 85 were individually interviewed, and 50 were diagnosed as 'cases'. 'Cases' consisted of 29 with definite or probable Gilles de la Tourette syndrome, 17 with definite or probable chronic multiple tics, and four with definite or probable obsessive–compulsive behaviour. Only eight of the 50 'cases' had consulted a doctor for their symptoms. 'Cases' and 'non-cases' could be distinguished on the basis of echo-phenomena, obsessive–compulsive features, self-injurious behaviour, and the trait score of the Leyton Obsessional Inventory, but did not differ significantly on any other psychopathological variables. The pattern of inheritance of the Gilles de la Tourette syndrome in this pedigree is consistent with autosomal dominant transmission.

The precise aetiology of the Gilles de la Tourette syndrome (GTS) is still unknown. In 1885, George Gilles de la Tourette postulated the hereditary nature of the disorder. Many investigators have subsequently demonstrated that GTS and chronic multiple tics (CMT) show a familial pattern (Eldridge *et al*, 1977; Golden, 1978; Guggenheim, 1979; Kidd *et al*, 1980; Nee *et al*, 1980; Pauls *et al*, 1981; Jagger *et al*, 1982; Erenberg *et al*, 1986; Lacey, 1986). Moreover, data from studies involving 11 concordant monozygotic GTS sets of twins suggest a genetic influence in GTS (Escalar *et al*, 1972; Wasman *et al*, 1978; Shapiro *et al*, 1978; Jenkins & Ashby, 1983; Waserman *et al*, 1983; Vieregge, 1987) and CMT (Price *et al*, 1985). In the latter study 43 pairs of same-sex twins were studied in which at least one co-twin had GTS. Concordance rates for GTS were 53% for monozygotic pairs and 8% for dizygotic pairs, but when diagnostic criteria were broadened to include tics in a co-twin, concordance rates rose to 77% and 23% for monozygotic and dizygotic twins respectively. It has been suggested that in families with GTS, CMT may be a milder manifestation of the disease (Pauls *et al*, 1981). A study of one large Mennonite extended family, of whom 159 were interviewed, suggests the transmission of GTS and CMT is autosomal dominant (Kurlan *et al*, 1986, 1987).

Recent psychopathological studies suggest that obsessive–compulsive behaviour (OCB) is not only severe in many patients with GTS (Frankel *et al*, 1986), but may be an integral part of the disorder, as it is significantly associated with central features of the syndrome such as copro- and echo-phenomena (Robertson *et al*, 1988). This link between GTS and OCB does not appear to be culturally determined, as OCB has been reported as occurring in significant proportions of American GTS cohorts (Comings & Comings, 1987*a*,*b*), and in equivalent cross-cultural American–British cohorts (Frankel *et al*, 1986), and in both British (Robertson *et al*, 1988) and Dutch (van der Wetering *et al*, 1988) GTS populations. Moreover, recent studies have suggested that GTS and OCB may be aetiologically and even genetically related (Pauls *et al*, 1986*a*,*b*).

Initially identified through the index case who presented at the clinic at the National Hospital for Nervous Diseases, Queen Square, we have identified and studied a large British kindred spanning six generations, and including 122 members plus spouses. In this paper we report the clinical and psychopathological features in the members of this extended family.

Method

We evaluated individuals in the pedigree over a two-year period. Initially, the subjects were interviewed to assess 'caseness', using a short, semistructured interview, lasting approximately 15–20 minutes, and devised by one of us (MMR) who is well acquainted with the clinical presentation and diagnosis of GTS. 'Caseness' was defined as the presence of symptoms or history sufficient to fulfil the criteria of one of the five diagnostic categories (see below). In addition, each individual over 18 years-of-age was assessed using the Schedule for Affective Disorders and Schizophrenia, Lifetime version (SADS–L; Endicott, 1978). The adult members of the pedigree (over 18 years) were also asked to complete the following standardised psychiatric rating scales to assess specific aspects of psychopathology: the questionnaire form of the Leyton Obsessional Inventory (Snowdon, 1980); the Crown-Crisp Experiential Index (CCEI), previously known as the Middlesex Hospital Questionnaire (Crown & Crisp, 1966); the Hostility and Direction of Hostility Questionnaire (HDHQ; Caine *et al*, 1967); and the 30-item version of the General Health Questionnaire (GHQ; Goldberg, 1972). These particular rating scales were chosen as it has been reported that patients with GTS referred to a clinic not only have high general psychopathology, but that both obsessionality and hostility are intimately linked to central features of GTS (Robertson *et al*, 1988).

Fifty-eight pedigree members were reinterviewed, using a more detailed questionnaire (taking one to two hours to complete) about symptoms and behaviour recognised in patients with GTS including the following: the presence and types of motor and vocal tics; the age of onset of symptoms; the severity of the tics; suppressibility and duration of the symptoms. The presence of associated behaviour such as coprolalia, copropraxia, echolalia, echopraxia, palilalia, palipraxia, stammering, stuttering, self-injurious impulses, the urge of feeling forced to touch, aggressive behaviour, and sleep disturbances were also inquired after. In addition obsessional thoughts, compulsions and rituals, and other behaviour which may be considered to be obsessional, such as arithmomania and 'evening up', were assessed. Further historical information was obtained by direct interview with reliable informants.

Individuals were assigned to diagnostic categories as follows:

(a) definite GTS: satisfying DSM-III (American Psychiatric Association, 1980) criteria on history and examination
(b) probable GTS: symptoms observed but no history obtained, or history obtained but no symptoms observed
(c) definite CMT: DSM-III diagnosis on history and examination
(d) probable CMT: as for probable GTS but without vocalisations
(e) Obsessive-compulsive behaviour: obtained on history and, in the case of children, corroborated by parents.

Global illness severity was rated according to the following:

(a) mild: present but not requiring medication and not interfering with social life
(b) moderate: present and requiring medication
(c) severe: as for (b) but severely socially disabling. Severely socially disabling in this context means that an individual is unable to live a normal life with respect to employment, interpersonal relationships and living independently.

Statistical analysis

The data were analysed using the χ^2 test and Fisher's exact probability test for contingency tables. The Mann-Whitney *U*-test was used for examining the relationships between

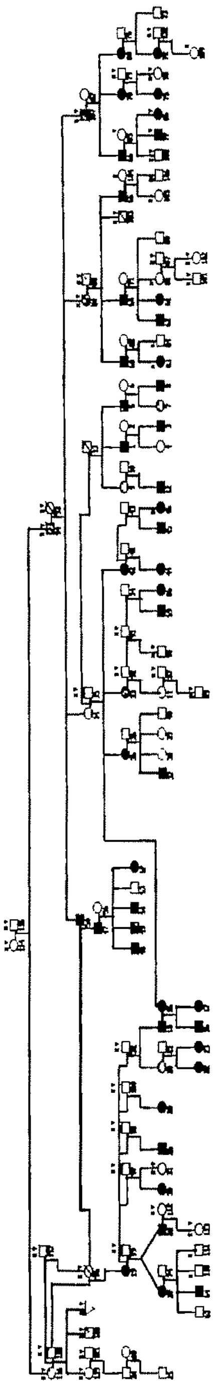

Fig. 1 Pedigree of family with Gilles de la Tourette syndrome (□ man, ○ woman, ● GTS and probable GTS, ● CMT and probable CMT, ⊕ OCB and probable OCB, X no blood taken, Y not interviewed, Ø deceased).

discrete and continuous variables. Non-parametric tests were employed because the continuous variables were not normally distributed. The data were analysed on the EUCLID computer at University College London, using the MINITAB and EASISTAT statistical packages.

Results

Of 122 members of a family multiply affected by GTS, 85 were interviewed at least once. Fifty-eight were interviewed twice. Six were seen and diagnosed as 'non-cases': five of these were too young for meaningful diagnosis, and one adult, while happy to be seen, was unwilling to answer detailed questions. The family, including members who were not interviewed, are shown in Fig. 1. Information about 31 of these was obtained from other family members, and considered to be sufficient for diagnosis (hence the fact that there are more than the 50 'cases' in the genogram), but not for the analysis of detailed psychopathological data. Information about the remaining six was considered unreliable for the purpose of this communication.

Statistical analysis was therefore performed on 79 individuals about whom there was adequate information. Of the 85 subjects interviewed, 50 were diagnosed as 'cases', as shown in Table I. Cases 27 and 28 were dizygotic twins concordant for GTS. The mean age of the 'cases' was 31.9 years (range 5–84) and that of 'non-cases' was 36 years (range 6–81). Age at onset of symptoms (tics or obsessive-compulsive symptoms) was 8.6±4.6 years (mean+s.d.). For the 50 'cases' (24 men, 26 women), eight had consulted a doctor for an opinion about their symptoms: two for tics; two for a cough; three for behavioural problems, and the index case for GTS at the clinic at the National Hospital for Nervous Diseases, Queen Square. Of these eight, three received treatment from psychiatrists; only one was given drug treatment, following a diagnosis of schizophrenia. Of the 50 'cases' graded for severity, one was graded as severe, one was moderate (index case), and the remaining 48 were mild. For these 50 subjects, associated behaviour was elicited (Table II). Twenty-nine subjects were diagnosed as having definite or probable GTS (Table I). The associated behaviour of these 29 GTS patients is shown in Table III.

TABLE I

Diagnoses of affected members

Diagnosis	*No. of individuals*
Definite GTS without OCB	18
Definite GTS with OCB	1
Probable GTS without OCB	9
Probable GTS with OCB	1
Definite CMT without OCB	7
Definite CMT with OCB	2
Probable CMT without OCB	6
Probable CMT with OCB	2
OCB	3
Probable OCB	1
Total	50

GTS, Gilles de la Tourette Syndrome, CMT, chronic motor tic, OCB, obsessive–compulsive behaviour.

The results of the standardised rating scales are presented in Table IV. No significant differences were found in scores between 'cases' and 'non-cases', with the exception of the trait score of the Leyton Obsessional Inventory, which was significantly higher for the 'cases'. When the scores for the 29 GTS 'cases' only were compared with 'non-cases', no significant differences emerged.

Fifty-two subjects were assessed using the SADS-L, and diagnoses are shown in Table V. Seven subjects received more than one diagnosis. Of the 21 subjects to whom

TABLE II

Associated behaviours and features of 50 'cases'

Associated behaviour/ feature	*No. of 'cases'*	*No. of 'non-cases'*	*Significance between 'cases' and 'non-cases'*^{1}
Sleep disturbance	32	11	NS
'Evening up'	16	0	$P=0.0001$
Forced to touch	11	0	$P=0.0035$
Echolalia	10	0	$P=0.0055$
Aggressive behaviour	10	1	NS
Arithmomania	8	0	$P=0.019$
Echopraxia	8	0	$P=0.0174$
Self-injurious behaviour	6	0	NS
History of stuttering	4	1	NS
Mental coprolalia	3	0	NS
Coprolalia	1	0	NS
Copropraxia	1	0	NS
Palipraxia	1	0	NS
Palilalia	0	0	NS

1. P= Fisher's exact probability test.

TABLE III

Associated behaviour and features of 29 definite or probable GTS 'cases'

Associated behaviour/ feature	*No. of 'cases'*	*No. of 'non-cases'*	*Significance between 'cases' and 'non-cases'*^{1}
Sleep disturbance	17	11	NS
'Evening up'	6	0	$P=0.012$
Forced to touch	7	0	$P=0.0065$
Echolalia	7	0	$P=0.0054$
Aggressive behaviour	6	1	NS
Arithmomania	3	0	NS
Echopraxia	7	0	$P=0.0054$
Self-injurious behaviour	5	0	$P=0.038$
History of stuttering	4	1	NS
Mental coprolalia	1	0	NS
Coprolalia	1	0	NS
Copropraxia	1	0	NS
Palipraxia	1	0	NS
Palilalia	0	0	NS

1. P= Fisher's exact probability test.

Table IV
Psychopathology assessed by rating scales

Rating scale	*'Cases'* *mean*	*s.d.*	*'Non-cases'* *mean*	*s.d.*	*Significance*[1]
Leyton Obsessional Inventory					
Symptom	13.13	8.1	9.86	7.3	NS Trait
	8.90	3.2	7.00	4.1	$U = 1216$, $P = 0.04$
Total	22.03	10.4	16.86	10.6	NS
Crown Crisp Experiential Index					
Free floating anxiety	5.14	4.2	5.65	4.6	NS
Phobic anxiety	4.62	3.8	4.80	2.7	NS
Somatic anxiety	4.95	3.5	4.25	3.0	NS
Depression	4.41	3.3	4.40	3.7	NS
Hysteria	5.08	3.4	5.15	2.8	NS
Obsessional	5.68	3.2	5.40	2.5	NS
Total	29.86	16.9	29.65	14.3	NS
General Health Questionnaire	4.28	5.9	3.45	5.9	NS
Hostility and Direction of Hostility Questionnaire					
Total	16.61	8.7	14.95	6.4	NS
Direction	−1.03	6.02	−0.84	4.6	NS

1. Mann–Whitney U-test.

Table V
Psychopathology assessed by SADS–L

Diagnosis	*No. of cases*
Major depressive disorder	10
Alcohol abuse	4
Hypomania episode	3
Generalised anxiety disorder	2
Cyclothymic personality disorder	2
Labile personality disorder	2
Schizophrenia	2
Phobic disorder	1

SADS–L diagnoses were assigned, 15 were 'cases', while six were 'non-cases' ($\chi^2 = 0.59$; 1 d.f; NS). As there are so few case reports on GTS and schizophrenia coexisting, we briefly present our two cases, though the association may be a chance finding.

Case reports

Case 1

Miss A is 23 years old and single, living with her parents. She was first seen by a psychiatrist in 1986, aged 22, with a presenting complaint of disturbed behaviour, particularly violence. At the time it was noted that she experienced visual and auditory hallucinations, the latter in the second person. Also noted were obsessive–compulsive phenomena, with repeating of the middle letters of words. The initial formulation was that she had a severe personality disorder with "intense ambivalent feelings". A psychotic illness could not be excluded. She was therefore treated initially with oral neuroleptics, then depot long-acting neuroleptics. At interview in late 1987, however, there was evidence of Schneiderian first-rank symptoms, namely auditory hallucinations in the third person, and passivity, with made actions. At that time, when we interviewed her, she admitted to marked obsessive–compulsive phenomena (arithmomania, 'evening-up', intrusive thoughts, and compulsions to move objects), in addition to severe motor and vocal tics (including animal noises), together with coprolalia, copropraxia, echolalia, echopraxia, and palilalia. Her nuclear family corroborated the history of GTS dating back to 12 years of age. This combination of symptoms resulted in severe social disability. She lived at home with her parents, was unemployed, and had no friends.

Case 2

Mrs B, a 58-year-old, separated woman, has had a long history of psychiatric illness since the early 1960s, following the birth of her eighth child. She has warranted at least six in-patient admissions and prolonged attendance at a day hospital, necessitating treatment for many years for schizophrenia with intramuscular flupenthixol decanoate, and oral neuroleptics when required. She currently resides in a community-based psychiatric residential hostel. When interviewed by us for the study she spoke of "Mr Clock", who has spoken to her since 1962, commenting on her actions in the third person. She has also experienced other auditory hallucinations; for example when the traffic is busy she hears a certain man and his associates gossiping about her. These voices are usually intrusive and derogatory. In addition, she experiences thought broadcasting and passivity. There was also evidence of several negative symptoms of schizophrenia such as blunted affect, avolition, and apathy. On direct questioning Mrs B admitted to the following: finger drumming, looking at her watch constantly without wanting to know the time, and looking in mirrors – all as 'habits'. At interview she cleared her throat several times, and admitted to that and coughing, both as 'habits'. She admitted to arithmomania, in that she had many counting obsessions and had done so since the age of 16. She also admitted to obsessional rituals embracing dressing and undressing, bathing and 'evening-up', in that she felt the need to have symmetry and order. This had begun in her 20s and has continued to date. She was unsure of the age of onset of other GTS symptoms, but they had been present as long as she could remember. It must be noted that she has been on dopamine antagonists for many years for her schizophrenia, which may have masked other motor or vocal tics.

Of note is that three of the bipolar affectives (type II, DSM-III-R; American Psychiatric Association, 1987) are her children: we suggest this is probably as a consequence of assortative mating. Attempts were made to contact the fathers of the children, but letters and telephone calls were consistently unanswered.

Blood samples

Blood samples were collected from 77 family members for DNA extraction. Analysis of linkage with a large series of restriction fragment-length polymorphisms (RFLPs) markers and mini-satellite mapping is in progress. Segregation analysis is also in progress and will be reported in a future communication.

Discussion

Our findings in a large pedigree of many affected individuals, the majority of whom have never sought or received medical attention, would support previous suggestions from both pedigree (Kurlan *et al*, 1987) and epidemiological (Caine *et al*, 1988), data that GTS in the community is more common than previously estimated, usually mild and thus often undiagnosed. Moreover, the frequency of 'caseness' in our pedigree is strikingly similar to that found by Kurlan *et al* (1987), with 54 'cases' in their pedigree of 159 members. The only difference from the Kurlan study is that we included as 'cases' subjects with OCB, but as can be seen from Table I, there were only four such individuals with OCB as a sole diagnosis. We would point out that our number of 'cases' may be an underestimate, as nine who were under the age of 21 were interviewed and labelled 'non-cases', but some of these individuals may well develop GTS at a later age.

Sleep disturbance, which has been documented widely in GTS (Robertson, 1989), was the most common of the associated behaviours, occurring in 32 of our 'cases', but this was not significantly more common than in 'non-cases'. It is noteworthy that 'evening-up' was not only the next most common associated behaviour, but also a significant discriminator between 'cases' and 'non-cases'. Arithmomania, another obsessional characteristic, was also significantly commoner in 'cases' than 'non-cases', as was the feeling or compulsion of being forced to touch. The only other behaviours discriminating between 'cases' and 'non-cases' were echolalia and echopraxia, known to occur in roughly 30–40% of GTS populations (Robertson, 1989) and widely recognised as being integral parts of the syndrome in the initial paper by George Gilles de la Tourette himself in 1885. When the associated behaviours of only GTS and probably GTS 'cases' were compared with 'non-cases', arithmomania failed to reach significance, but self-injurious behaviour was found to discriminate significantly between the two groups. This is particularly noteworthy as we (Robertson *et al*, 1989) have reported self-injury as occurring in one-third of a cohort of 90 GTS patients, suggesting that self-injury in the GTS population has been under-reported to date.

It is important to address the issue as to how typical our 'cases' were in their GTS symptomatology when compared with GTS subjects in other investigations. Our patients exhibited most commonly a variety of OCB, such as 'evening up', arithmomania, and the feeling of being forced to touch, which discriminated between 'cases' and 'non-cases'. This is typical of a substantial proportion of GTS individuals encountered in a wide variety of settings, including clinical (Frankel *et al*, 1986; Robertson *et al*, 1988) and epidemiological (Caine *et al*, 1988) cohorts, as well as pedigree/family studies (Kurlan *et al*, 1986). Thus, our findings suggest our cases are typical and in keeping with the literature, which adds to the argument that OCB is an integral part of the syndrome (Robertson, 1989), irrespective of the severity of GTS symptoms. Sleep disturbance has been reported to be common in GTS subjects, independent of cohort setting, namely epidemiological (Caine *et al*, 1988) or clinical (Robertson *et al*, 1988); it is of importance in this context that it has been argued that GTS is a disorder of arousal (Glaze *et al*, 1982; Barabas *et al*, 1984*a*,*b*, 1985), although there have been opponents to this view (Erenberg, 1985). Other associated behaviours seen in smaller numbers of our pedigree cases were aggression, self-injurious behaviour and, in only one case, copro-phenomena. It would seem that our cases are not unusual in that relatively small numbers of sufferers exhibited these behaviours, and although such behaviours are documented in patients with GTS, they are more common in those patients who have been referred to physicians and may well be a reflection of the severity of the disorder, and thus a result of ascertainment or referral bias; for the majority of GTS subjects in the community, it is a mild disorder and thus often undiagnosed (Robertson, 1989), and this was the situation with the majority of our cases.

The trait scores of the Leyton Obsessional Inventory were the only ones to discriminate between 'cases' and 'non-cases', thus arguing again for an intimate association between OCB and GTS. Thus, although a significant association between obsessive-compulsive phenomena and core features such as coprophenomena and echo-phenomena has only been documented once (Robertson *et al*, 1988), there is a growing literature on obsessive-compulsive disorder and GTS (Frankel *et al*, 1986; Comings & Comings, 1985; Hagin *et al*, 1982; Montgomery *et al*, 1982; Nee *et al*, 1980; Green & Pitman, 1986).

With regard to the general psychopathology, it can be seen from Table IV that there are no differences in the scores of the rating scales between 'cases' and 'non-cases'. Moreover, the results of the SADS-L show 21 family members had had a psychiatric illness during their lifetime. When doing a breakdown of the specific diagnoses, these are not in excess of the lifetime risks for schizophrenia (1%), major depressive disorder (20%), alcohol abuse (13%), and the others, which could be subsumed under the diagnosis 'neurosis', which has a lifetime risk of 13% (American Psychiatric Association, 1980; Kendell & Zealley, 1983). Moreover, two schizophrenics in a cohort of 122 people is not above the expected (Kendell & Zealley, 1983) and hence our findings support the assertion that there is no connection between schizophrenia and GTS (Robertson, 1989), as has been reflected by only a few case reports in the literature (Caine *et al*, 1978). Our data of depressive disorders, schizophrenia, phobic states, and generalised anxiety not being more common in the GTS population than in the general population does not support the suggestion that these pathologies and GTS are genetically related (Comings *et al*, 1984).

The pattern of inheritance of GTS and CMT in our pedigree supports an autosomal dominant pattern of inheritance, as suggested by previous authors (Devor, 1984; Price *et al*, 1984; Pauls & Leckman, 1986; Kurlan *et al*, 1986, 1987) but this is only evident when the presumed phenotype is broadened to include CMT and OCB. Even when this is done, penetrance is apparently incomplete, as there are two unaffected individuals who, if paternity has been correctly detemined, are obligate carriers of the disease gene (nos 116 and 96). However, proof that GTS, CMT, and OCB are genetically related will have to await the demonstration of linkage between GTS and polymorphic genetic markers. The pedigree is a suitable one for linkage analysis because it is unlikely that heterogeneity of linkage is present. However, due account for the presence of the two inbreeding loops (cases 18 married to 15, and also 96 to 50) will have to be taken into account in the calculation of lod scores.

Conclusions

Our pedigree data confirm those of previous investigators suggesting that the majority of GTS cases in the community are mild and go undetected: thus the true prevalence of GTS remains unknown. Our data also support the notion that GTS and obsessionality are intimately related. Likewise, echophenomena are an integral part of the syndrome. We further suggest that self-injurious behaviour occurs in a clinically significant proportion of GTS patients, that it often goes undetected, and that it can occur in those individuals who have mild GTS symptoms. Finally, inspection of the pattern of affected individuals within the family suggests autosomal dominant inheritance when the presumed phenotype is broadened to include CMT and OCB.

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Psychopathology in Institutionalised Mentally Retarded Adults

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The clients in an institution for the mentally retarded were screened for signs of psychopathology with the Psychopathology Instrument for Mentally Retarded Adults (PIMRA). Of the 168 clients, 146 satisfied criteria for DSM–III axis I disorders, and 153 satisfied criteria for at least one disorder on either axis I or II. Multiple diagnoses were common, and a total of 288 diagnoses were found. No particular disorder was found to be more prevalent in those with organic brain dysfunction. The level of functioning was not found to predispose to any particular diagnosis. There was a positive relationship between the presence of a psychiatric disorder and the resources available to the clients and the activities offered. The prevalence of psychiatric disorders was extremely high in the population studied, with the exception of the affective disorders, of which no case was found. One possible reason for this high prevalence is that deinstitutionalisation leaves behind those with the more severe behavioural problems.

There has been a long-standing debate concerning the occurrence and causes of psychiatric disorders among the mentally retarded since Esquirol in 1828 claimed that mental retardation and psychiatric disorders were separate entities (Hayman, 1939). Phelps (1897) held that mental illness and mental retardation were part of a continuum and could not be entirely separated. In contrast, Tredgold (1908) claimed that mental disorders of the same kind as seen in the normal population were found in the mentally retarded, but the latter group was much more frequently afflicted. Kraepelin held an intermediate position and suggested that some cases of dementia praecox developed as a result of mental retardation, and that some cases of mental retardation resulted from an early onset of psychosis.

Later works have largely confirmed that psychiatric disorders are separate entities which occur more commonly among the mentally retarded. Dewan's (1948) results indicate an inverse relationship between IQ and emotional instability. A high prevalence of behavioural problems/psychiatric disorders has been established both in institutionalised populations and among those living in community residential facilities or private homes (Intagliata & Rinck, 1985; Hill *et al*, 1985). Epidemiological approaches like Lund's (1985) and Göstason's (1985) confirm a high frequency of these problems. Gillberg *et al* (1986) and McQueen *et al* (1987) show that this tendency manifests itself in youth. Day's (1985) results regarding the middle-aged and elderly mentally retarded indicate that an increased susceptibility to psychiatric disorder continues into old age. This is consistent with Eyman & Widaman's (1987) findings.

In spite of a rich literature in this field, the prevalence of different forms of psychopathology remains unclear. There are several reasons for this. Studies have involved different populations in respect of age, degree of retardation, and living conditions. The first two factors seem to influence both frequency and type of behavioural disturbances, while living conditions are likely to have a complex interaction with the presence and type of behavioural disturbance, as well as with contemporary political views and economic factors. Additionally, there is no consensus regarding diagnostic criteria for psychiatric disorders in the mentally retarded. The emergence of DSM–III (American Psychiatric Association, 1980) has to some degree ameliorated this situation. Prevailing opinion suggests that these criteria are useful and mostly valid for mildly and moderately mentally retarded subjects, but considerable doubt exists concerning the more severely handicapped (Reid, 1983). Sovner (1986) reviewed the limitations of DSM–III for mentally retarded persons and indicated some adjustment for degree of mental handicap.

Surveys addressing the use of drugs among the mentally retarded show high frequencies of psychotropic drug use (Lipman, 1986), particularly neuroleptics. One would expect that these prescription practices reflect the psychiatric condition of the users.

The present study is concerned with the distribution and frequency of psychiatric disorders in an institution for the mentally retarded. A specially adapted version of the DSM–III criteria, the Psychopathology Instrument for Mentally Retarded Adults (PIMRA; Senatore *et al*, 1985), was used.

Method

Population

Of the total 221 persons living in the institution, 168 fulfilled the inclusion criteria of age (16–65 years).

The institution consists of 27 separate wards, most of them in separate buildings, with 3–12 (mean 8.28) clients on each. This population was established in the 1950s, when mentally retarded people were generally cared for in large, centralised institutions. Since the late 1960s efforts have been made to transfer clients to smaller, community-based settings and home care. This has resulted in the number of clients in 1987 being about a third of that in the late 1960s. It is reasonable to assume that the population under study is heavily selected and is unrepresentative of mentally retarded persons in general.

Data collection

The head of each ward filled in a questionnaire regarding the general resources available to the ward. One of the ward personnel in regular contact with the client for at least six months completed three forms. One described demographic variables, the kind and amount of activities the client participated in, and various kinds of behavioural disturbances. Another described the level of functional handicap in various aspects of daily activities, and a global evaluation of mental health. The same member of staff also filled in the informant version of the PIMRA. We used the mental retardation classification of ICD–9 (World Health Organization, 1978), which parallels the DSM–III classification. The data from the PIMRA were analysed to give a total score, subscale scores, and an indication as to whether DSM–III criteria for each diagnostic group were satisfied.

Information about medical health and weight was obtained from the case records.

Information about the general population was obtained from the Norwegian Central Bureau of Statistics (1987).

Instruments

The PIMRA is based on DSM–III and consists of 56 items in eight subscales; schizophrenia, affective disorder, psychosexual disorder, adjustment disorder, anxiety disorder, somatoform disorder, personality disorder, and poor mental adjustment. Aman *et al* (1986*a*) found good inter-rater reliability ($r=0.95$–0.97) with the self-report version of the PIMRA. The internal consistency of subscales varied between 0.45 and 0.73 (mean 0.64). Stability of ratings over time was low. The effects of age, sex, functional level, and living conditions were found to be small. The original presentation of the scale (Senatore *et al*, 1985) found the informant version's internal consistency equivalent to a coefficient $\alpha=0.83$. The test–retest correlations were significant for all subscales, and varied between 0.48 and 1.00 when retested after approximately 22 weeks. The depression subscale, which was more extensively studied by Kazdin *et al* (1983), correlated significantly with the Hamilton Rating Scale for Depression (informant version of PIMRA), and with the Beck Depression Inventory (self-rating version of PIMRA).

Informed consent

Consent for the study was obtained from the regional ethical committee for medical research, the owner of the institution, each client's legally responsible caretaker, the bureau for supervision of the Norwegian data protection act, and the Norwegian Department for Health and Social Welfare.

Results

The PIMRA was properly completed for 164 clients (97.6%). The mean age of these clients was 40.4 years. The age distributions were similar for the catchment area population and the 128 clients from this area. The male : female ratio was 2 : 1. Distribution across levels of retardation was as follows: 15.2% were profoundly, 50.9% severely, 20.1% moderately, and 3.6% mildly retarded. It was not possible to classify 9.7% owing to their multiple handicaps. There was an insignificant tendency for the women to be more retarded than the men.

Fifteen clients were scored on the PIMRA by two raters, and single-item inter-rater reliability corresponded to $\kappa=0.64$ (85.7% agreement). The number of positive diagnoses in each category was too small to compute inter-rater reliability for diagnostic categories.

The composition of the diagnostic categories is given in Table I. Only schizophrenia, affective disorder, anxiety disorder, adjustment disorder, and no axis I diagnosis were considered mutually exclusive categories. Somatoform, psychosexual, and personality disorder were considered compatible with existing axis I diagnosis, except for schizophrenia. The total number of diagnoses was greater than the total number of clients, with a mean of 1.76 diagnoses per person.

According to DSM–III, the existence of organic brain dysfunction makes use of the functional major diagnostic categories dubious, as it is difficult to assess whether the disturbance is caused by the dysfunction. The role of brain dysfunction is unclear for most psychiatric conditions. The proportion of the diagnoses coexisting with obvious signs of brain dysfunction (epilepsy or cerebral palsy) was 30.6%. This proportion did not differ significantly between the diagnostic categories.

The profoundly retarded group has a significantly lower proportion of persons with psychiatric disorders ($P<0.001$). Other variations in diagnoses across different levels of retardation are not significant.

The three largest mutually exclusive groups, schizophrenia, anxiety, and no diagnosis on axis I, were subjected to one-way analyses of variance, with subsequent Tukey procedures. The variables analysed were: number of clients on the ward, ward size (m^2), number of trained nurses on the ward, total number of personnel on the ward, number of hours the client was actively engaged per week, global mental health score by the informant, and client's age.

TABLE I
Diagnostic groups by degree of mental retardation (n = 164)

Diagnostic category	*Degree of retardation*										
	Profound (n = 25)		*Severe* (n = 84)		*Moderate* (n = 33)		*Mild* (n = 6)		*Uncertain* (n = 16)		*Totals*
	no.	*%*	*no.*	*%*	*no.*	*%*	*no.*	*%*	*no.*	*%*	*no.*
Schizophrenia	0	0.0	34	70.8	9	18.7	1	2.1	4	8.3	48
Affective disorder	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0
Adjustment disorder	2	50.0	0	0.0	0	0.0	0	0.0	2	50.0	4
Anxiety disorder	12	12.7	44	46.8	24	25.5	4	4.3	10	10.6	94
Somatoform disorder	0	0.0	4	40.0	4	40.0	1	10.0	1	10.0	10
Psychosexual disorder	0	0.0	7	38.8	8	44.4	1	5.6	2	11.1	18
Personality disorder	14	16.4	38	44.7	21	24.7	2	2.4	10	11.7	85
No axis I disorder	11	61.1*	4	22.2	1	5.6	0	0.0	2	11.1	18
No axis I or II disorder	8	72.7*	2	18.1	0	0.0	0	0.0	1	9.0	11

$P<0.001$ (deviation from expected distribution of mental level for this diagnostic category).

The 'no diagnosis' group achieved a better score on global mental health evaluation, the number of hours they were actively engaged per week was approximately half of that seen in the other two groups, and they also lived in more crowded wards ($P<0.05$). Differences between the two diagnostic groups and for the other variables were not significant.

Discussion

According to the prevalence figures of Göstason (1985), one would expect, in total, 788 mildly to moderately retarded, and 655 severely or profoundly retarded persons in the catchment area of the institution. From this area there were 32 mildly/moderately retarded and 82 profoundly/severely retarded persons living in the institution (4.1% and 12.5% of the expected total numbers). This indicates a narrow selection of clients, which would be expected to be skewed towards an over-representation of those needing more care. This may explain the high frequency of psychiatric problems found in this study.

The most surprising finding in diagnostic distribution was the low frequency of affective disorder, which is among the more common psychiatric disorders in the normal population. Eaton & Menolascino (1982), Russel & Tanguay (1981), Lund (1985), and Gillberg *et al* (1986) also found a low frequency of affective disorders. Others, including Myers (1986), Benson (1985), and Reiss (1982), have found frequencies of affective disorders more closely resembling those of the rest of the population. Sovner & Hurley (1983), in their review of the literature on affective disorder among the mentally retarded, leave little doubt that the classic affective syndromes exist in this population. The various settings and populations studied explain some of the variation in results, and affective disorders do seem to be less common among the mentally retarded, particularly the most retarded, than others.

Our inability to discover clients with affective disorders may be because we sought only a current episode of major depression – being a time-limited disorder, only a small proportion of those cases would have been identified, and the diagnostic criteria excluded less serious depressions. Furthermore, the population studied was dominated by severely and profoundly retarded people.

The schizophrenia category probably includes persons with other psychoses, which may account for some of the 48 diagnoses of schizophrenia given. However, it remains a surprisingly high number. The diagnosis of schizophrenia in the PIMRA is based on active behaviour and symptoms, the mere possession of autistic traits being insufficient for this diagnosis.

A high frequency of anxiety syndromes was found. Other authors have found both a low (Lund, 1985) and a high (Ollendick & Ollendick, 1982) frequency of neurotic problems among the mentally retarded. This might be due to differences in diagnostic criteria and population selection.

The low frequency of problem behaviour or psychiatric diagnoses in the profoundly retarded group is thought to be due to the inability of these individuals to display the types of behaviour necessary to fulfil the PIMRA diagnostic criteria. We suspect the PIMRA to be inadequate as a diagnostic tool for these groups. An alternative possibility is that the psychiatric categories as defined in DSM-III are inadequate or even irrelevant for these persons, as suggested by Reid (1983).

We have provided the figures for personality disorders as indicated by the PIMRA, but have little confidence in their validity. We feel that the instrument is inadequate for these diagnoses in its present form, since diagnostic criteria are based on single-item responses together with a high score on the poor mental adjustment subscale. Most clients with personality disorder diagnoses satisfied the PIMRA criteria for more than one category of personality disorder.

The PIMRA does not diagnose infantile autism. From the case records it was found that ten of the clients had a diagnosis of pervasive developmental disorder of early or late onset. Five of these fulfilled the PIMRA criteria for schizophrenia.

A further group of diagnostic categories lacking in the PIMRA are the organic psychiatric syndromes. It is possible to regroup our client population on the basis of gross signs of cerebral dysfunction (epilepsy or cerebral palsy). On this basis, the group with cerebral dysfunction constitutes 26.2% of the total axis-I diagnoses, being the second largest group after anxiety disorder. This is compatible with earlier studies (e.g. Göstason, 1985).

Analysis of variance did not indicate that the degree of behavioural problems and psychiatric disturbance were related to lack of stimuli or resources: where there was unequal distribution of resources between healthy and behaviourally disturbed clients, the most disturbed lived on less crowded wards and were offered more activity.

This study has confirmed most of the major findings from previous reports of psychiatric disorder among the mentally retarded. The proportion of clients suffering from behavioural disturbances compatible with diagnostic criteria for psychiatric disorders is much higher than that found in population-based epidemiological surveys (Lund, 1985; McQueen *et al*, 1987). This confirms our impression that deinstitutionalisation has left the 'problem clients' in the old institutions. This is probably due to a failure of community-based care to provide for them. It is an indication that present strategies for giving such individuals a life outside the large institutions are insufficient. There is need for more knowledge and research efforts.

Acknowledgements, references, and authors' details are given at the end of the following paper, on p. 530.

British Journal of Psychiatry, (1990), **156**, 525–530

Frequency of and Determinants for Psychotropic Drug Use in an Institution for the Mentally Retarded

OLAV M. LINAKER

The frequency of psychotropic and anticonvulsant drug use in 168 institutionalised mentally retarded adults was studied. Use of neuroleptics and anticonvulsants was more frequent and use of hypnotics and antidepressants less frequent than in the general population. Neuroleptics were given to 49% of the population. Clients with no psychiatric diagnosis consumed less neuroleptics than those with such a diagnosis, and there was a non-significant trend for those with a more serious diagnosis (e.g. schizophrenia) to take a higher dosage. The degree of disruptive behaviour and the availability of a physician were related to dosage of neuroleptics. The various psychiatric diagnoses given could explain only a small proportion of the variance in dosage.

According to Intagliata & Rinck (1985), the use of psychotropic medication among the mentally retarded is widespread. Surveys from the United States indicate that 40–50% of residents in institutions for the mentally retarded take psychotropic drugs, compared with 25–35% of the mentally retarded in community settings. Comparable figures have been found in other countries (Göstason, 1985; Lund, 1986;

Reid & Ballinger, 1987). Most authors found evidence of increasing frequency of psychotropic drug use with greater degree of mental handicap, and Aman (1984) considered the mentally retarded to be one of the most medicated groups in society.

Psychotropic medication should generally be given as treatment for a specific psychiatric symptom or diagnosis. The drug choice, dosage, method of administration, and duration of treatment should be decided upon from experience with the condition and the treatment. The decision to treat behavioural disturbances should be made with due consideration of the possible side-effects of drugs and the possibility of using treatment modes other than (or in addition to) drugs. On these premises drugs have great potential in alleviating some behavioural and emotional disturbances.

The high frequency of psychotropic drug use among the mentally retarded has raised serious questions regarding whether the above principles are followed. It now seems to be agreed that psychiatric disorders are more prevalent among the mentally retarded than in the rest of the population, but classification of behavioural disturbances in this group is still controversial, especially among the most retarded. Sovner (1986) criticised the use of unmodified diagnostic criteria for psychiatric disorders (DSM-III, American Psychiatric Association, 1980) in mentally retarded populations. Reid (1983) and Gualtieri & Keppel (1985) suggest that ordinary criteria may be valid for the mildly and moderately retarded, but doubt their validity in the more severely handicapped.

In addition, the natural development of behavioural disturbances among the mentally retarded is little studied. Eyman & Widaman (1987) found only small changes in adaptive behaviour over four years in their longitudinal study, indicating that many of these problems are of long duration. Details about which problems last how long in which persons in given surroundings are sparse.

Few drugs have been thoroughly studied in relation to effects, side-effects, and effective dosage when taken by mentally retarded persons, and the lack of well conducted outcome studies unfortunately also holds true for most alternative treatment modes. During recent years there has been a growing criticism of the use of psychotropic drugs for behavioural problems among the mentally retarded. Aman & Singh (1986) review the arguments and advise that although they find final judgement premature, the criticism should be taken seriously, and researchers are generally urged to improve documentation of both beneficial and adverse effects.

In all studies of the mentally retarded, the neuroleptics are reported to be the most prevalent psychotropic drug group. The neuroleptics have a range of side-effects which one would expect to be especially harmful for mentally retarded, such as interference with motor function and learning, and sedation. The tendency to produce such effects varies with dosage and different neuroleptics. Neuroleptics also carry the risk of permanent damage, in the form of tardive dyskinesia (Gualtieri *et al*, 1986) and the life-threatening malignant neuroleptic syndrome. The advent of law suits for long-term side-effects has made the problem of documenting existing practices or the change of these even more pressing.

The present study was motivated by the experience of entering an institution for the mentally retarded as a psychiatric adviser. It set out to determine the extent of psychotropic drug use and factors contributing to this kind of treatment.

Method

The population under study, diagnostic procedures, etc., are described in the previous paper (this issue, pp. 522–525). Informed consent and data handling procedures are the same in this study. Information about client medication was obtained from the central register of the institution, in which all medication (type and dosage) was noted. For dosages of neuroleptics, anti-Parkinsonian drugs, sedatives, hypnotics, antidepressants, and anticonvulsants, a transformation to a daily dosage measure was performed. The actual daily dosage was calculated as a percentage of defined daily dosages (DDD). The standards for grouping and for defined daily dosage were those of the Anatomical Therapeutic Chemical Classification (ATC), accepted by the World Health Organization Drug Utilization Research Group (World Health Organization, 1979) for international studies of drug consumption. One DDD corresponds to 300 mg of chlorpromazine, 10 mg of diazepam, 5 mg of nitrazepame, 100 mg of imipramine, 25 mg of procyclidine, or 1 g of carbamazepine.

Information about drug consumption in the general population of Norway was obtained from the National Bureau for Drug Control (Norsk Medisinaldepot, 1986). These levels of medication are not adjusted for age. The age distribution of the sample did not differ significantly from that of the general population.

Results

A total of 136 (81.0%) of the inhabitants of the institution received daily medication. Some kind of medical diagnosis which could justify continuous or periodical treatment was present in 147 (87.5%). Psychotropic medication was taken regularly by 82 (48.8%) (by 95 (56.5%) if one includes the anticonvulsants).

There was a small but significant correlation with age for the sedatives ($r = 0.17$, $p = 0.034$). The neuroleptic group

TABLE I

Number of defined daily dosages per 1000 inhabitants per day

Drug group	*In general population*[1]	*In study population*	P[2]
Neuroleptics	9.9	376.2	0.000
Sedatives	23.6	27.5	0.586
Hypnotics	35.7	22.8	0.026
Antidepressants	8.9	3.0	0.042
Anti-Parkinsonian	0.7	15.6	0.000
Anticonvulsants	6.8	293.8	0.000

1. Total Norway 1985. Not adjusted for age.
2. Comparisons made with χ^2 tests, two-tailed P values.

showed a significant positive correlation with intelligence ($r=0.18$, $P=0.025$), and the anticonvulsants had a significant negative correlation with the same variable ($r=-0.19$, $P=0.024$). The other drug groups did not show significant correlations.

Differences in dosage between the sexes were not significant, although all drugs except anticonvulsants had a higher mean dosage in males.

The number of DDDs/1000 inhabitants/day for each group of psychotropic drugs is given for the study population and for the general population in Table I. Table II shows drug use by axis I diagnostic categories. It also shows the average dose, given in percentage of DDD, for the actual drug users in each drug class. There were no significant differences in frequency of drug use between diagnostic categories for any drug group.

One-way analysis of variance with subsequent Tukey tests between the schizophrenia, anxiety disorder, and no axis I disorder groups ($n=159$) was performed for the dosage level of the various medication groups. The results showed no difference between groups for anti-Parkinsonian drugs, sedatives, hypnotics, antidepressants, or anticonvulsants. For the neuroleptics there was a significant difference ($F(2,156)=4.44$, $P=0.013$) between the no axis I disorder group and the two other groups, but not between schizophrenia and anxiety disorders. A tendency for higher dosage in the schizophrenia group was suggested by a mean dosage of 70.2% of DDD versus 28.9% of DDD in the anxiety group.

One group ($n=10$) which according to medical records had been given the diagnosis autistic disorder of either infancy or childhood onset, used an average 124.2% of DDD. Five of these satisfied the PIMRA schizophrenia criteria and had an average dose of 220.2 of DDD, in contrast with the average dosage of 28.2% of DDD used by the remaining five clients.

Of the users of neuroleptics, about half used doses less than 50% of what is considered the average antipsychotic dose (corresponding to 300 mg chlorpromazine). About 25% of these low-dosage users were in the schizophrenia group. Sixteen per cent of the users had a dosage of more than 150% of the DDD. Of these high-dosage users, 73% were in the schizophrenia group. The average dose in chlorpromazine equivalents corresponds to 2.06 mg/kg for the total sample, 4.56 mg/kg for those satisfying criteria for a diagnosis of schizophrenia, and 6.37 mg/kg for those of the schizophrenic clients who were actually taking neuroleptics.

A multiple-regression analysis of determinants for neuroleptic total dosage included 33 variables: neuroleptic dosage (% of DDD), PIMRA diagnosis of schizophrenia, total PIMRA score, score on PIMRA schizophrenic disorder subscale, score on PIMRA affective disorder subscale, score on PIMRA psychosexual disorder subscale, score on PIMRA adjustment disorder subscale, score on PIMRA anxiety disorder subscale, score on PIMRA somatoform disorder subscale, score on PIMRA personality disorder subscale, score on PIMRA poor mental adjustment subscale, number of clients on each ward, space on each ward, number of special nurses (or equivalent) in each ward, number of personnel on each ward, number of personnel per client in each ward, availability of psychologist, availability of physician, availability of educational service, availability of special educational services, activity offered systematically (hours per week), communicative ability, degree of mental retardation (ICD-9/DSM-III classes), raw score for degree of mental retardation, physical health (ill/not ill), ward personnel evaluation of mental health, age, weight, aggressive behaviour towards people in last year (yes/no), self-mutilative behaviour in last year (yes/no), destructive behaviour in last year (yes/no), other disruptive

TABLE II

Frequency of use for each drug group for the different axis I diagnostic groups and for the total study population

Drug group	*Schizophrenia* (n=*48)*		*Anxiety disorders* (n=*94)*		*Adjustment disorders* (n=*4)*		*No diagnosis* (n=*18)*		*Total* (n=*163)*[2]	
	%	*AU*[1]	%	*AU*	%	*AU*	%	*AU*	%	*AU*
Neuroleptics	48	147	38	75	50	38	39	22	42	92
Anti-Parkinsonian	2	60	2	100	0	0	0	0	2	87
Sedatives	4	100	3	87	0	0	0	0	3	92
Hypnotics	6	93	1	100	0	0	0	0	2	87
Antidepressant	2	25	1	25	0	0	0	0	1	25
Antiepileptics	19	159	19	130	25	120	33	133	21	138

1. AU, average dosage (%DDD) for those actually using drugs.
2. Medication data missing for one client.

TABLE III

Results from the stepwise multiple-regression analysis for neuroleptic dosage (n = 167)

Independent variable	*Beta*	*Simple* r	*Accumulative* R^2	*Multiple* R
Disruptive behaviour	0.26	0.33	0.14	0.37
Physician available	0.35	0.47	0.20	0.45
Somatisation disorder subscale score	0.19	0.33	0.25	0.50
Weight	0.25	0.25	0.28	0.53
Activity offered	−0.28	−0.08	0.30	0.55
No. of clients in ward	−0.25	−0.03	0.34	0.59
Communicative ability	−0.20	−0.12	0.37	0.61
Soundness subscale score	−0.14	−0.17	0.38	0.62

Note: $F(8,159) = 12.29$, $P < 0.001$. Criterion for entering equation was set to $P < 0.05$. Missing values were substituted with variable means.

behaviour in last year (yes/no), disruptive behaviour (sum of scores on the last four variables).

These variables can be grouped in the following main categories: diagnosis of schizophrenia, scores on PIMRA with subscales, personal space, nursing and staff resources, client actively engaged, characteristics of client, and different forms of disruptive behaviour. A stepwise procedure was followed. Missing data were substituted with variable means. Probability of F-to-enter equation was set to 0.05 and probability of F-to-leave equation to 0.1. Eight variables were selected by the procedure, giving a total R^2 of 0.38, and a multiple R of 0.62 ($P < 0.001$), as shown in Table III.

Table III shows that the availability of a physician is the factor closest correlated with dose of neuroleptics, followed by disruptive behaviour. Of the disruptive behaviour subvariables, the tendency to destroy property had the highest correlation with neuroleptic dosage, followed by aggression towards people. Somatic complaints without medical explanation also contributed to dosage variations. Not unexpectedly, weight is positively correlated with dosage. There is a slight tendency for lower dosage with increasing number of hours of active engagement, more clients on the ward, decreasing communicative ability, and high scores on the soundness subscale of the PIMRA.

In a second multiple-regression analysis only the eight subscales of the PIMRA were included, with neuroleptic dosage as the dependent variable. Only the scores on the adjustment disorder scale and the affective disorder scale showed significant covariance with dosage ($F < 0.001$), but the proportion of total variance explained by these factors was small ($R^2 = 0.12$). The sample was divided into two, with a median split for functional intelligence level, and two separate multiple-regression analyses of PIMRA subscales with neuroleptic dosage as the dependent variable were performed. These showed that the subscale scores gave $R^2 = 0.07$ for the lowest functional level, and $R^2 = 0.18$ for the less retarded half of the sample. This indicates that psychiatric diagnosis is a more important determinant for neuroleptic drug use in the less retarded persons.

Discussion

This study was performed in a population with an over-representation of the profoundly and severely retarded. There was also a higher prevalence of psychiatric disorders than in epidemiologically selected populations. The results confirm that mentally retarded are given psychotropic medication with a frequency which we would find alarming in any other group, except psychiatric in-patients. The medications given are predominantly neuroleptics, which in fact accounted for virtually all of the relative excess.

Psychiatric diagnosis did not seem to be the main reason for administration of psychotropic medication, whether for the less retarded or the most disabled. The proportion of the variance in dosage explained by diagnostic subgroups in the two separate regression equations is small in both, although somewhat higher for the less retarded. The degree of disruptive behavioural problems seems to play a more important role. This kind of behaviour may or may not be due to psychiatric disorder; in itself it is not commonly regarded as a reason for medication. The practice of giving neuroleptics was not observed to be significantly influenced by the type of psychiatric diagnosis, although there were significant differences between those with a diagnosis and those without. There was also a non-significant trend for non-psychotic clients to receive smaller doses than those satisfying criteria for schizophrenia. These findings are consistent with earlier findings regarding the influence of diagnosis on medication (Matson *et al*, 1984; Hill *et al*, 1985).

The high frequency of psychiatric disorders/behavioural problems among the mentally retarded (previous paper, this issue, pp. 522–525) leads us to expect a somewhat increased frequency of medication in this population. It is however doubtful whether this can justify the enormous increase in neuroleptic use (Hill *et al*, 1985). The best treatment strategies for different forms of behavioural disturbances in the mentally retarded are still to be demonstrated. The lack of data does not allow us to state that the apparent widespread use of medication is unsound.

During the last five years it has been the policy of the medical administration at the institution to reduce or eliminate psychotropic drugs whenever possible. However, an investigation at the same institution ten years earlier, when the population was 50% higher, showed exactly the same frequency (49%) of psychotropic drug use (Petersen, 1978). It may be that neuroleptics are a help for mentally retarded subjects suffering from behavioural disturbances in general, or this may be true for only some conditions met in this population. However, clinical experience in normal populations would not suggest this drug group to be a panacea for behavioural problems.

We also found a high prevalence of anxiety disorder, which led us to expect a high frequency of drugs commonly used in the treatment of these conditions. This was not confirmed. The results from this population and other studies (Hill *et al*, 1985) do not show that the anxiety disorders are treated psychopharmacologically in a manner which would normally be considered adequate. To the contrary, the results indicate suboptimal treatment of anxiety disorders in the mentally retarded. Aman (1983) considers sedatives to be contraindicated in the mentally retarded, but the use of such agents to treat anxiety in this population has not been much investigated (Lipman, 1986). The same holds true for the antidepressants, which have recently shown an interesting potential for treatment of various anxiety disorders (Aman *et al*, 1986*b*).

The fact that the variable with the highest correlation with neuroleptics dosage in the multiple-regression analysis was the availability of a physician may be an indication that physicians are too quick to administer drugs when behavioural problems occur. Mouchka (1985) points to the potential danger in medicalising behaviour, and stresses the fact that one's view of where the root of a problem lies will influence the way one tries to solve it. However, the study design does not exclude the opposite conclusion: that the physicians of the institution are adequately distributed, according to need. This reasoning also applies to the other variables selected in the multiple-regression analysis. We cannot be sure whether the results imply a causal relationship or simply covariance due to other reasons not included in the analysis. One would like to be optimistic, and interpret the apparently negative effect of activity offered on the neuroleptic dosage as indicating that if the clients are offered more activity the need for medication will be reduced. Owing to the design of this study, the reasons for this covariance are obscure, and an equally valid explanation may be that clients with less disturbing behaviour are easier to activate and are also given less medication.

The shortcomings of the study should be borne in mind when interpreting the results, which should be regarded as helpful indices for the ethically and clinically necessary further study of treatment strategies for the psychiatric disorders among the mentally retarded.

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Cancer and Mental Handicap
A Further Study (1976–85)

J. JANCAR

In four hospitals for the mentally handicapped, 302 patients died during the decade 1976–85; of these, 53 (17.5%) died from cancer. The percentage of deaths from all types of cancer during that period showed an increase, particularly in gastrointestinal cancer, as compared with the previous four decades

Jancar & Jancar (1977) reported a survey of cancer in the mentally handicapped who died in the Stoke Park group of hospitals in Bristol. The 40-year survey (1936–75) revealed a marked increase in the number of deaths from cancer during the last two decades (1956–75), particularly from cancer of the gastrointestinal tract. The average age at which both male and female patients died from cancer was 56.5 years, but the average age of death during the last five years (1971–75) was double the average age during the first five years (1936–40).

Carter & Jancar (1983) reported a very detailed study of mortality in the mentally handicapped during 50 years (1930–80) of the same Stoke Park hospitals. There had been a marked change in the causes of death during this period. While tuberculosis was no longer a major cause, other terminal respiratory tract infections were still prevalent. Deaths due to status epilepticus had decreased, with a concomitant increase in those due to carcinoma, myocardial infarction, and cerebrovascular accident. During the latter 25 years the longevity in patients with Down's syndrome increased on average by 40 years and in other mentally handicapped patients by more than 30 years.

In view of the continuing increased incidence of cancer and increased longevity of the patients as revealed in these two reports, it was decided to study a further decade (1976–85) and to compare the results with previous findings.

Method

Between 1909 and 1948 patients were admitted to the Stoke Park group of hospitals from all counties of England and Wales, and from the south-west of England between 1948 and 1974. Since then the hospitals have served a health district with a population of approximately 220 000. The patient population averaged 1600 during the two previous surveys, with approximately equal distribution of the sexes.

Because of the changes of admission and discharge policy in recent years, the hospital population is gradually decreasing. In the present study the population in 1980 dropped to 1240 patients and in 1984 to 1051 (555 males and 496 females) (Fig. 1).

Diagnostic details concerning the deaths from cancer of the mentally handicapped were obtained from the death register and individual case files containing the history, diagnosis, results of investigations, and post-mortem when available.

Results

The total number of deaths during the period 1976–85 was 302 (173 women, 129 men), of whom 53 (17.5%) (34 women, 19 men) died from cancer. The percentage of deaths for all types of cancer showed a marked increase compared with the previous four decades (Fig. 2).

Table I shows the various types of cancer in the 53 patients.

The average age at death from carcinoma in the 1976 study was 56.5 years. In the present study (1976–80) it had risen to 65 years. However, over 1981–85 the average age at death rose further, to 74 years in women and 68.8 years in men.

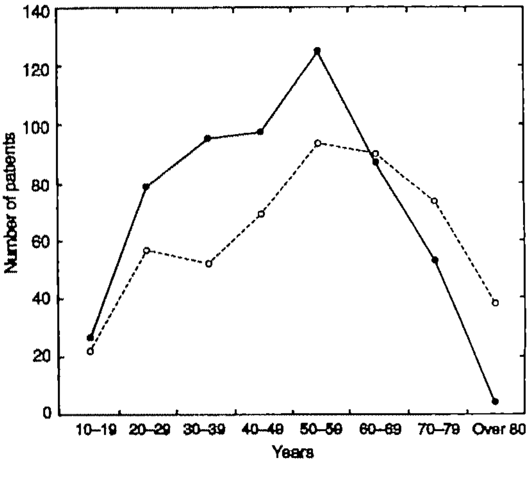

Fig. 1 Hospital population in four hospitals in 1984 divided into decades (●——● men, $n=555$; ○——○ women, $n=496$)

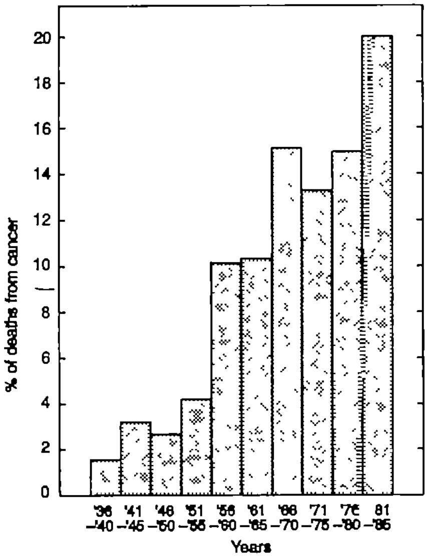

FIG. 2 The percentage of deaths from all types of cancer (both sexes) over five decades.

TABLE I
Site of cancer in 53 terminal cases

		Women	*Men*
Gastrointestinal tract	31 (58.5%)	22 (41.5%)	9 (17.0%)
Breast	5	5	—
Gall-bladder	4	2	2
Liver	4	2	2
Uterus	2	2	—
Bladder	2	—	2
Bronchus	2	—	2
Kidney	1	1	—
Thymus	1	—	1
Hodgkin's disease	1	—	1
Total	53	34	19

The patients' IQs

The patients who died from cancer had frequent intellectual assessments during their lifetime. The IQs in the present study ranged from 15 to 56. The average IQ for the women was 37.4, which was significantly higher than the average for the men, which was 30, whereas in the 1977 study the average IQ for women was 43 and for men 41. It appears that with the increasing lifespan of severely mentally handicapped patients the average IQ of the patients is falling.

Epilepsy

In this study 34 mentally handicapped patients with epilepsy (20 women, 14 men) died, but only two female and no male epileptics died from cancer. One had cancer of the uterus and another cancer of the oesophagus. In the 1977 study there were five women and three men who died from cancer, but none from cancer of the liver. It is interesting to note that all epileptics in the four hospitals were on anticonvulsant drugs for many years, sometimes in high dosages, and none developed cancer of the liver.

White *et al* (1979) studied 636 epileptic patients who died in the Chalfont Centre for Epilepsy, of whom 78 died from cancer. They reported that "Apart from the brain and central nervous system, no particular site had a significant excess of tumours. In particular, there were no liver tumours (and only one gallbladder carcinoma). This makes it unlikely that the liver tumours produced on feeding phenobarbitone to mice are indicators of major human risk."

Down's syndrome

In the 1976–85 sample, 24 patients suffering from Down's syndrome died. Two of the 11 women were under 40 years of age and nine over 40 years (average age 55.4 years). Five of the 13 men were under 40 years of age and eight were over 40 years (average age 57.6 years). None of the Down's syndrome patients died from cancer.

Of the 115 patients suffering from Down's syndrome who died in our hospitals during 1936–75, only one man with mongoloid features died from cancer of the stomach and one woman died of acute lymphoblastic leukaemia.

One Down's syndrome patient with left testicular malignant teratoma, who is still alive, has been successfully surgically treated.

Discussion

Fifty years ago patient mortality was considerably higher in all age groups of the mentally handicapped than in the general population. This difference is now relatively small. These changes have been most marked during the past 30 years because of better medical care, introduction of new antibiotics and anticonvulsant drugs, as well as better diet, general care, and environment for the mentally handicapped population.

The increased longevity is associated with additional mental and physical disorders and diseases, including carcinomas, and the effects of ageing with new problems for the patients, the families, carers, and planners. Day (1987) and McLoughlin (1988) also found increased longevity and its associated problems and diseases in their mental handicap hospitals.

Although the present study of cancer includes only a selected sample of the hospitalised mentally handicapped population for which no control group is available, the results do highlight a number of points which may be helpful for research into causes and prevention of cancer. The findings have also an important implication for the planning of services for the ageing mentally handicapped population in hospital and in the community alike.

The findings of an increased incidence of cancer, in particular gastrointestinal cancer, may reflect factors other than increasing longevity alone. For example there is an increasing amount of evidence to suggest a relationship between low serum cholesterol levels and an increased incidence of cancer of the colon. We studied 23 patients (12 men, 11 women) who died from cancer of the colon (Jancar *et al*, 1984). Serum cholesterol levels in both sexes were significantly lower in these patients compared with those who died from other causes ($P<0.01$). In men, serum cholesterol levels were also significantly lower in those who died from cancer of the colon in comparison with those who died from other forms of cancer ($P<0.01$). Isles *et al* (1989) reported the same inverse relation, in the normal population, between plasma cholesterol and both cancer and other causes of death.

The number of patients who died from cancer of the gastrointestinal tract, particularly women, continued to rise. The most frequently affected organs were the stomach (five women, six men), followed by oesophagus (five women, two men), rectum (five women) and colon (four women).

Cancer of the breast occurred in five women. In the 1977 study 13 women died from breast cancer.

The incidence of cancer of the bronchus (two men) is small in spite of the number of patients who smoked, some very heavily. A low incidence was also noticed in the 1977 study (two men, one woman).

None of the 24 Down's syndrome patients in the survey died from cancer.

Early detection of cancer in the mentally handicapped population is often difficult. There are communication problems, particularly with the severely mentally handicapped. Associated syndromes with neurological and other disorders make the diagnosis more difficult and the threshold of pain often differs from that of the normal population. The following case illustrates these problems.

A woman with the features of Cockayne's syndrome and an IQ of 43 developed at the age of 60 a swelling in the right side of the neck. Aspiration biopsy revealed the presence of malignant cells, possibly of squamous origin. On further examination the primary source of cancer could not be detected. Palliative radiotherapy was given with an initially good response. However, her general condition began to deteriorate and she died from bronchopneumonia at the age of 62. Post-mortem revealed the primary tumour to be squamous carcinoma of the posterior third of the tongue with metastases in the cervical lymph nodes. Neurohistopathological examination also confirmed the Cockayne's syndrome.

The aetiology of many forms of cancer in this study remains obscure. The increased longevity is an obvious factor contributing to the increase in some cancer mortality rates within this sample. Diet, drugs, genetics, biochemical and biophysical factors have also to be taken into account when researching causes of cancer.

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The Psychiatric Status of Patients with the Chronic Fatigue Syndrome

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The prevalence of psychiatric disorder in 48 patients with chronic fatigue syndrome (CFS) was determined. Twenty-two had had a major depressive (non-endogenous) episode during the course of their illness, while seven had a current major (non-endogenous) depression. The pre-morbid prevalence of major depression (12.5%) and of total psychiatric disorder (24.5%) was no higher than general community estimates. The pattern of psychiatric symptoms in the CFS patients was significantly different to that of 48 patients with non-endogenous depression, but was comparable with that observed in other medical disorders. Patients with CFS were not excessively hypochrondriacal. We conclude that psychological disturbance is likely to be a consequence of, rather than an antecedent risk factor to the syndrome.

> "Acquired neurasthenia is characterised by a diminished power of attention, distractibility, defective mental application, difficulty of thinking, an increased susceptibility to fatigue, increased emotional irritability, and a greater variety of physical symptoms, mostly subjective, including hypochondriasis." (Emil Kraepelin, 1981, first published 1907)

The chronic fatigue syndrome (also termed 'myalgic encephalomyelitis', 'Royal Free disease', 'post-viral fatigue syndrome'), recently defined by the Centers for Disease Control in Atlanta, Georgia (Holmes *et al*, 1988), has been subject to ongoing controversy (Dawson, 1987; David *et al*, 1988; Swartz, 1988). Psychological features have often been noted (David *et al*, 1988), leading some physicians to regard CFS as a psychiatric disorder (McEvedy & Beard, 1973). The demonstration of persistent infection (Yousef *et al*, 1988) and immunological abnormalities (Behan *et al*, 1985; Straus *et al*, 1985; Caliguiri *et al*, 1987; Lloyd *et al*, 1989), however, have supported alternative hypotheses (e.g. that of Wakefield & Lloyd, 1987) that highlight the role of biological factors in the condition.

Straus (1988) stated that "ultimately, any hypothesis regarding the cause of the chronic fatigue syndrome must incorporate the psychopathology that accompanies and, in some cases, precedes it," emphasising the importance of assessing psychopathology both pre-morbidly and during the illness.

The frequency of neuropsychiatric and depressive symptoms in these patients (David *et al*, 1988; Straus, 1988) suggested the specific hypothesis that CFS occurs in individuals with a pre-morbid vulnerability to depression. A case-control study of 24 subjects by Taerk *et al* (1987) supported this notion but may be challenged as controls were healthy hospital personnel, and no attempt was made to validate prevalence estimates. Similarly, Kruesi *et al* (1989) have reported an increased lifetime prevalence of depressive and anxiety disorders in patients with CFS.

In this paper we record the prevalence of psychiatric morbidity in patients with CFS both during and before the illness episode to determine whether the syndrome is associated with an increase in psychological disturbance, and whether subjects had a higher pre-morbid rate of psychiatric disturbance. We included a control group of patients with non-endogenous depression to assess whether CFS patients resembled patients with depression, either during the illness episode or on pre-morbid measures.

The non-specific nature of the key symptom of fatigue has necessitated the development of strict criteria for CFS (Holmes *et al*, 1988; Lloyd *et al*, 1988*a*,*b*). The Centers for Disease Control (CDC) criteria (Holmes *et al*, 1988) specifically exclude patients with a number of psychiatric disorders and those taking psychotropic medication, effectively preventing accurate quantification of psychological symptoms in patients with CFS. The difficulty created by those criteria is exemplified by the study of Manu *et al* (1988), who reported that 95% of 135 consecutive patients who had attended a fatigue clinic in a university hospital, with at least a six-month history of debilitating fatigue, failed to meet CDC criteria. The overwhelming reason for exclusion was current psychiatric disorder (91/135) and the most prevalent psychiatric diagnosis was "major depression" (67/91). Such concurrent psychiatric morbidity was assumed to be "a *major cause* of these patients' fatigue" (italics added). The criteria used in the present study, however, do not make this assumption, and thereby avoid the limitation of excluding subjects on the basis of psychological symptoms.

Method

We studied 48 subjects with marked and persistent fatigue who met the following operational criteria for CFS (Lloyd *et al*, 1988*a*):

> To fulfil the criteria a patient must have chronic, persistent or relapsing fatigue of a generalised nature, causing major disruption of usual daily activities, present for more than six months, plus two major *or* one major and three minor criteria (symptoms, signs, or assessments):
>
> (a) Symptoms: persisting at least six months continuously, or relapsing on three or more occasions with a similar pattern over six months or more:
>   (i) Major: concentration/memory impairment
>   (ii) Minor: myalgia, arthralgia, depression, tinnitus, paraesthesia, headaches.
>
> (b) Signs: present on at least one occasion subsequent to the initial illness:
>   (i) Major: lymphadenopathy
>   (ii) Minor: pharyngitis, muscle tenderness.
>
> (c) Immunological assessment:
>   (i) Major: cutaneous anergy, T4 or T8 lymphopenia
>   (ii) Minor: hypoergy

The criteria that we have recommended since are a simplification of those original criteria (Lloyd *et al*, 1988*b*). Both of our sets of criteria emphasise the importance of a characteristic pattern of physical and neuropsychological impairment and the demonstration of impaired cell-mediated immunity.

All patients had been referred by their general practitioner to the immunology or infectious diseases' department of a university teaching hospital for evaluation of their chronic fatigue. Patients referred to this service typically had been extensively investigated by their own practitioners and/or other consultant physicians. Additionally, many had been assessed previously by a psychiatrist. The patients in this report were drawn from a previously described sample of 200 patients (Lloyd *et al*, 1988*b*) who had been assessed rigorously to exclude other possible physical causes of fatigue (infectious diseases or thyroid, neurological, haematological, hepatic, renal, or autoimmune dysfunction). Although the CDC criteria were not established at the time our patients were enrolled, the process of physical evaluation and investigation we used was as extensive as that recommended by Holmes *et al* (1988).

Twenty-nine of the 48 patients had developed their persistent fatigue immediately after an acute infectious illness. Five of these illnesses had been documented serologically (four Epstein–Barr virus infections, one toxoplasmosis). Patients were approached to progress to a treatment trial on the basis of the persistence of their symptoms and their willingness to give informed consent to participate in a double-blind, placebo-controlled, treatment trial of intravenous immunoglobulin therapy (to be reported elsewhere). The extensive process of physical evaluation within a tertiary referral service, the application of strict clinical and laboratory criteria, and the delay between initial contact with the clinic and inclusion in the treatment trial, all limited the clinical heterogeneity that one might expect within such a sample.

Initially, each subject was asked by the psychiatrist (IH) to describe their 'key symptoms'. The interviewer recorded the frequency with which psychological and neuropsychological symptoms were reported spontaneously before administering a standardised psychiatric interview schedule (SCID–P; Spitzer & Williams, 1985) which generated DSM–III–R (American Psychiatric Association, 1987) diagnostic data. A relative of each patient was interviewed independently to corroborate the patient's report. Because of a delay in the commencement of the psychiatrist's involvement in the trial, 33 patients were interviewed before treatment, and the remaining 15 patients after completion of immunoglobulin infusions.

All subjects completed a number of psychometric measures, including the 30-item General Health Questionnaire (GHQ; Goldberg, 1979), the Zung Self-Rating Depression Scale (Zung, 1965), the Eysenck Personality Inventory (Eysenck & Eysenck, 1964), and the Illness Behaviour Questionnaire (Pilowsky & Spence, 1983). The latter scale generates scores on seven dimensions which indicate the subject's attitudes towards the illness. The 17-item Hamilton Rating Scale for Depression (Hamilton, 1960), and the Newcastle Depression Scale (Carney *et al*, 1965), used to identify patients with endogenous depression, were administered by the psychiatrist.

Forty-eight patients with non-endogenous depression (Newcastle Depression Scale score less than five) were selected from the in-patient and out-patient psychiatric services of the same hospital for comparison with the patients with CFS.

Results

The mean age of the patients was 36 (range 15–63) years, and there were equal numbers of men and women. The mean duration of illness was 56 (median 46, range 12–156) months. The sample therefore contained patients with chronic illness with little prospect of spontaneous remission.

The control subjects suffering from non-endogenous depression were matched exactly for sex, and similarly for age (36.1 v. 37.8 years, $t = 1.56$, NS) and social class (mean Congalton (1969) score: 3.69 v. 3.89, $t = 1.01$, NS). Of the 48 control patients, 28 met DSM–III–R criteria for major depression, while the remaining 20 had minor depressive disorders. None of these control patients had been depressed continuously for two years, so that there were no cases of dysthymic disorder. Nineteen were in-patients of a general hospital psychiatry unit, while 29 were from out-patient and liaison psychiatry clinics.

Of the 48 patients with CFS, 19 spontaneously reported psychological and 29 reported neuropsychological complaints among their key symptoms (Table I).

Twenty-four warranted a psychiatric diagnosis during the course of their illness. The commonest disorder was major

TABLE I

Frequency of spontaneous reports of neuropsychological or psychological symptoms as 'key' features by patients with CFS (n = 48)

Spontaneous symptoms	*No. of patients*	*Percentage of patients*
Neuropsychological impairments		
concentration/attention	25	52%
short-term memory	13	27%
dysphasia	4	8%
planning tasks	2	4%
Total	29	60%
Psychological symptoms		
depression	18	38%
irritability	8	17%
anxiety/worry/tension	4	8%
Total	19	40%
Total (neuropsychological and/or psychological)	38	80%

TABLE II

Mean scores from the Zung Self-Rating Depression Scale (n = 33)

Item no.	*Mean*
Most frequently reported Zung items	
12 Not easy to do things	3.5
10 Tiredness	3.4
2 Worse in a.m.	2.8
11 Mind not clear	2.8
16 Indecisive	2.4
Least frequently reported Zung items	
19 Suicidal	1.1
7 Weight loss	1.2
3 Tearful	1.4
13 Restless	1.5
9 Palpitations	1.5

TABLE III

Numbers of patients and relatives reporting psychiatric morbidity before onset of CFS (n = 48), *and controls* (n = 48) *before onset of depression*

DSM-III-R	*Patients*		*Depressive controls*
	Self-report	*Relatives' report*	
Major depression	6 (12.5%)	6 (12.5%)	30 (62%)
Panic disorder	1 (2%)	1 (2%)	3 (6%)
Alcohol dependence	4 (8%)	4 (8%)	18 (38%)
Benzodiazepine dependence	—	—	5 (10%)
Psychotic episode	1 (2%)	1 (2%)	—
Total previous morbidity	12 (24.5%)	12 (24.5%)	43 (90%)

(non-endogenous) depression (8 men, 14 women). Six of these 22 depressed patients with CFS also experienced panic attacks, although these were frequent enough in only one case to justify an additional diagnosis of panic disorder. In addition, three non-depressed patients had experienced at least one panic attack, and two of these subjects met criteria for panic disorder. Only one patient, who also met criteria for major depression, fulfilled criteria for somatisation disorder (Briquet's hysteria). There were no cases of generalised anxiety disorder and, although a number of patients reported simple phobias, none was associated with avoidant behaviour leading to significant impairment of daily functioning.

The overall concordance of subject/witness reports was very high (kappa = 0.87), with relatives reporting a psychiatric diagnosis in 23 of the patients, strongly supporting the accuracy of our estimate. In the 33 patients assessed before treatment, the prevalence rate of psychiatric disorder in the preceding month was 21% (7/33), all having major (non-endogenous) depression.

The GHQ scores of the 33 subjects assessed immediately before treatment established that 15 had a score of five or above, generally suggestive of psychiatric morbidity (Goldberg, 1979). In patients with known medical disorders, however, significant physical symptoms inevitably lead to positive responses on more of the GHQ items. Zung depression scores for the 33 subjects assessed before treatment showed that 19 scored above the recommended cut-off point of 40 (Zung, 1972) for identifying mild depression in community settings. As with the GHQ, the raw Zung score is known to be significantly elevated in medical patients (Zung *et al*, 1983). It is possible to overcome this effect, in part, by comparing the number of patients and depressive controls assigned to at least the moderately depressed range (48 or above) on the Zung scale. Using this higher cut-off point, a highly significant difference ($\chi^2 = 22.06$, $P < 0.001$) remained between the number of patients with CFS (8/33) in comparison to the number of depressive controls (37/48). This established that depressive disorders of clinical severity were rare in the CFS sample. Table II shows patients' mean scores on the Zung items.

This distinction was supported by the significant differences between the mean scores of the CFS patients and depressive controls on the Hamilton (10.6 v. 19.0, $t = 8.92$, $P < 0.001$) and Zung (40.7 v. 55.2, $t = 7.67$, $P < 0.001$) scales. Importantly, patients with CFS also reported significantly lower levels of neuroticism (7.3 v. 15.8, $t = 7.71$, $P < 0.001$). This suggested that any psychiatric morbidity in the CFS sample was less severe, in addition to being less prevalent, than in depressed patients.

Table III records the patients', their relatives', and the depressives' estimates of prior psychological morbidity. Again there was substantial agreement between the patients' and relatives' estimates of overall pre-morbid psychiatric morbidity in the CFS group (kappa = 0.81). Thirty of the 48 depressive controls had had a previous episode that met

TABLE IV

Mean scores on dimensions of illness behaviour of patients with CFS as measured by the Illness Behaviour Questionnaire (IBQ)

IBQ subscale	*CFS patients (n = 44) mean (s.d.)*	*General practice[1] (n = 147) mean*	*Briquet's syndrome[2] (n = 17) mean*
General hypochondriasis (range = 0–9)	1.96 (1.9)	1.44	4.0
Disease conviction (range = 0–6)	4.25 (1.2)	1.59	4.0
Psychological v. somatic focusing (range = 0–5)	0.45 (0.8)	1.99	1.0
Affective inhibition (range = 0–5)	1.98 (1.7)	2.46	4.0
Dysphoria (range = 0–5)	2.00 (1.7)	2.31	5.0
Denial (range = 0–5)	3.43 (1.6)	2.93	2.0
Irritability (range = 0–6)	2.93 (1.8)	2.45	3.0

1. Pilowsky & Spence (1983).
2. Singer *et al* (1988).

DSM-III-R criteria for major depression, compared with 6 of the 48 CFS patients ($\chi^2 = 25.6$, $P < 0.001$).

The scores of the patients with CFS on the seven dimensions of the Illness Behaviour Questionnaire are shown in Table IV. Comparison of these data with previously reported Australian general practice samples (Pilowsky & Spence, 1983) and patients with Briquet's hysteria (Singer *et al*, 1988) suggests that the illness behaviour of patients with CFS is characterised by the strong conviction that they are physically ill (i.e. high scores on 'disease conviction': "an attitude of determined certainty as to the presence of disease, and a resistance to reassurance" (Pilowsky, 1988) and low scores on 'psychological v. somatic concern', which is said to be indicative of "somatic focusing" (Pilowsky, 1988)).

In contrast to patients with Briquet's hysteria, who typically present their psychological concerns in a somatic form, patients with CFS are not hypochondriacal and are not inhibited in their expression of affectively laden topics. (A high score on 'affective inhibition' is said to indicate "a reluctance to communicate inner feelings to others, particularly those of hostility" and is felt to be related to the notions of "introspection and . . . alexithymia" (Pilowsky, 1988)). Patients with CFS do differ from general practice patients, however, principally in terms of their reluctance to accept psychological interpretations of their somatic symptoms. Further, they have somewhat higher scores on the 'denial' subscale, which purports to measure "the tendency to regard illness as the sole problem, whose resolution would result in a circumstance devoid of difficulties" and "the tendency to displace attention from psychosocial stressors onto physical problems" (Pilowsky, 1988).

Discussion

Eighty per cent of patients with CFS spontaneously reported that neuropsychological and/or psychological difficulties were among their key complaints, confirming that such symptoms are common components of the CFS syndrome. Half had a psychiatric diagnosis (usually major depression) during the course of the illness, and 21% met criteria for major (non-endogenous) depression at the start of treatment.

The existence of significant psychological morbidity during the illness raises the question as to whether CFS is a mislabelled depressive disorder or a depressive equivalent. As early as 1907 Kraepelin noted that "acquired neurasthenia" needed to be carefully differentiated from melancholia. If CFS is a form of depressive disorder, a similar pattern of current symptoms and past psychiatric disorder might be expected in CFS patients to that observed in depressed psychiatric patients. Our findings do not support that proposition.

The estimated prevalence of pre-morbid lifetime psychiatric disorder in our CFS subjects (24%) is comparable with that reported in three sites of the Epidemiological Catchment Area Survey (ECA), where the lifetime prevalences of psychiatric disorder (excluding phobic disorders) were 24.9%, 23.9%, and 26.2% respectively (Robins *et al*, 1984). Previous large epidemiological studies have indicated a lifetime prevalence rate for major depression of 18–25% (Weissman *et al*, 1978; Reich *et al*, 1980; Bromet *et al*, 1986), larger than our estimate of a 12.5% pre-morbid prevalence rate of depression in CFS patients. Corroborative data from relatives were highly consistent with reports by the subjects and offer strong support for the validity of our data.

When compared with non-endogenously depressed controls, our CFS patients did not have a similar high pre-morbid rate of psychiatric disorder, were significantly less neurotic, and, when unwell, neither clinically resembled nor had the psychometric profile of non-endogenous depressive patients seen in psychiatric settings. The percentage of subjects with non-specific psychiatric symptoms as determined by the GHQ in our sample is consistent with previous findings in general medical (Goldberg & Blackwell, 1970; Maguire *et al*, 1974) and neurological settings (Bridges & Goldberg, 1984).

The interviewing psychiatrist was not blind to the clinical diagnosis of the patients with CFS or depressive disorders, and so it is important to consider whether a systematic bias may have been introduced in the evaluation of prevalence rates of pre-morbid psychiatric disorder. Given the important differences in the type of symptoms reported by patients with CFS as compared with patients with depressive disorders, it is doubtful that a truly 'blind' investigation is possible. We sought, however, to avoid any potential bias by using a highly structured interviewing technique. Further, we corroborated our data in the CFS patients with prevalence results reported by relatives, and we established high levels of agreement. Neither the interviewees nor the relatives were reluctant to report recent psychiatric morbidity. It is unlikely, therefore, that the low pre-morbid rates of disorder can be attributed to systematic under-recording of psychiatric morbidity. It is possible, however, that a recall bias may have been operating, such that past psychiatric morbidity was not remembered by either patients or their relatives as readily as that morbidity associated with their current protracted illness.

Our findings do not confirm those of Taerk *et al* (1987), who suggested a much higher pre-morbid risk of depressive disorder in patients with CFS. The patients in that study were not as extensively evaluated as the patients in this study or that of Kruesi *et al* (1989), so that it is likely that their sample was less homogeneous. In their study, however, if one compares the pre-morbid prevalence rate of major depression in the patients (12/24), rather than the total lifetime prevalence rates (67%, which includes the period of the current illness), with the lifetime prevalence rate in the 'healthy' controls (7/24), then there is only a non-significant trend ($\chi^2=2.2$, NS) towards higher affective disorder in the patient group. Further, the use of the criteria from the Diagnostic Interview Schedule (DIS) and DSM-III in that study produced high prevalence rates of all psychiatric disorders in both the patients and the 'healthy controls', suggesting that the actual threshold for psychiatric diagnosis was low.

Although Taerk *et al* (1987) did not suggest that depression alone was the cause of their subjects' illnesses, they did suggest that "sporadic neuromyasthenia may be the result of an organic illness in psychologically susceptible (i.e. premorbidly depressed) individuals". By contrast, our results suggest that CFS patients are no more psychologically disturbed before the onset of their illness than members of the general population. Taerk *et al* used DSM-III criteria for major depression, whereas we used the more strict criteria of DSM-III-R, and so we would expect lower prevalence rates for this disorder in our study.

Kruesi *et al* (1989) studied 28 patients who were said to have met CDC criteria for CFS. The patients were interviewed with the DIS to assess lifetime prevalence rates of psychiatric disorder. Patients in that study had been ill for a mean of 6.8 years and had been extensively investigated to exclude other possible disorders, thereby creating a sample quite similar to our own. Contrary to the actual CDC criteria, however, the presence of a current or past psychiatric diagnosis did not exclude those patients from participation in a treatment trial of acyclovir, an antiviral therapy. Lifetime depressive disorders were identified in 54% (15/28) of their patients (13 cases of major depression), while 21% (6/28) were currently depressed. These depressive diagnoses, however, were closely related in time to the onset or course of the fatigue syndrome. Simple phobia was reported as clinically significant in eight patients (29%), a remarkably high proportion. Only two of their patients met criteria for somatisation disorder, while other significant anxiety disorders were rare (one case of panic disorder and no cases of generalised anxiety disorder).

Examination of the pre-morbid prevalence rates suggested that eight of their patients had simple phobia, one had panic disorder/agoraphobia, and only two had a major depressive disorder. Attaching surprising importance, however, to the pre-morbid diagnoses of simple phobia, the authors concluded that "psychiatric disorders more often preceded the chronic fatigue than followed it". Robins *et al* (1984) have noted the high inter-site variation in the prevalence of the diagnosis of simple phobia using the DIS and DSM-III criteria, and reported their lifetime prevalence data for all disorders both with and without this diagnostic category. The high rate of simple phobia reported by Kruesi *et al* (1989) is therefore likely to be a result of the interview method and is unlikely to be of psychopathological significance in patients with CFS.

If one limits evaluation of their cases to those with axis I diagnoses, but excludes simple phobia, then there are important similarities between their findings and ours. The pre-morbid rate of psychiatric disorder was low, 18% (5/28) (one patient with agoraphobia, two with major depression, and two cases of alcohol abuse, allowing that each diagnosis was actually for a different individual), comparable with our premorbid rate of 25%. Depressive disorders were common in association with the fatigue syndrome (54% as compared with our rate of 46%), with a currently depressed case rate of 21%, which is identical to our figure.

Our method of case selection involved the enrolment of individuals who had been extensively evaluated within a tertiary referral service and who had often experienced considerable delay between confirmation of the diagnosis and participation in the treatment trial. This process may have decreased the rate of current psychiatric symptoms. No active psychiatric screening was undertaken by the hospital medical staff before inclusion in the trial, but our patients cannot be considered comparable with patients presenting for the first time complaining of fatigue in general medical settings. Establishing the pattern of pre-morbid and current psychopathology in a group of patients who clearly meet strict clinical and immunological criteria for the disorder is essential, however, before progressing to the evaluation of patients who may share only the symptom of fatigue in common.

The pattern of illness behaviour recorded in our patients was not similar to that previously described in patients with defined Briquet's hysteria (Singer *et al*, 1988), as patients with CFS were not particularly hypochondriacal, affectively inhibited, or dysphoric. If patients with CFS were dealing principally with psychological distress by the process of somatisation, one might have expected higher scores on those relevant subscales.

Our patients differed from those in Australian general practice, principally in terms of their conviction that they were physically ill. Given the controversy surrounding this disorder, the referral of these patients to a tertiary assessment service and their participation in a pharmacological treatment trial, the firmness with which they held this belief and rejected psychological interpretations of the cause of their illness is not surprising. It may be of considerable clinical importance, however, as conflict may quickly arise between such patients and medical practitioners who are openly sceptical about the disorder.

In conclusion, we have demonstrated that while depression and anxiety are common symptoms in patients suffering from CFS, there is no evidence from our well-defined sample to support the hypothesis that CFS is a somatic presentation of an underlying psychological disorder. In particular, there is no evidence that CFS is a variant or expression of a depressive disorder. Instead, our study supports the hypothesis that the current psychological symptoms of patients with CFS are a consequence of the disorder, rather than evidence of antecedent vulnerability. We are unable to say whether the psychological symptoms occur purely as a result of the fatigue, or as an integral symptom of the disease process. Comparisons between patients with CFS and patients who have fatigue on a peripheral basis (e.g. myasthenia gravis) or on a central basis (e.g. multiple sclerosis (Krupp *et al*, 1988)) will be undertaken to clarify this issue further.

It is possible that the chronic production of cytokines, such as interferon, which is released by lymphocytes in response to viral infections, may account for the morbidity reported by these patients. These soluble products of lymphocytes may precipitate fatigue and neuropsychiatric syndromes in humans (Denicoff *et al*, 1987; McDonald *et al*, 1987) and, therefore, ongoing localised production of these substances within the central nervous system of patients with CFS constitutes a possible explanation for the diverse physical, neuropsychiatric, and psychological symptoms encountered in this syndrome (Wakefield & Lloyd, 1987).

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The Management and Treatment of Personality-Disordered Patients
The Use of Sequential Diagrammatic Reformulation

HILARY BEARD, MARTIN MARLOWE and ANTHONY RYLE

The clinical management of patients with personality disorders is seldom satisfactory. It is suggested that the bewilderment provoked and experienced by these patients can be reduced by a careful analysis of their shifting states of mind. The construction of diagrams tracing such shifts is helpful to both patients and clinicians. Illustrative case histories are presented

Patients with personality disorders of the DSM-III borderline, histrionic, antisocial, and narcissistic varieties (American Psychiatric Association, 1980) are encountered in various settings, including accident and emergency departments after episodes of self-harm, probation departments, addiction treatment units, in forensic settings, and by social workers. In general psychiatry they are not popular. Psychiatrists are happy to treat the relatively common accompanying depression (Perry, 1985; Tarnapolsky & Berelowitz, 1987) but seldom have much to offer for the personality problems, for these patients are often uncooperative and disruptive. Some are referred for psychotherapy, but they are regarded as difficult (Waldinger, 1987). The reason for this unpopularity lies in the nature of the disorder; these patients are prone to express powerful, poorly controlled emotions, and to generate extreme reactions of both concern and rejection in staff, who find the inconsistency of their moods confusing, and who have difficulty in accepting their propensity to harm themselves and undermine attempts to help. These features have been seen by psychoanalysts (e.g. Kernberg, 1975) as reflecting an unstable personality, characterised by opposing or disconnected 'subpersonalities'; this is manifested in a tendency to polarise relations with others between idealised, undifferentiated attachment on the one hand, and bitter, conflicted, or abusive relationships on the other (in which the patient may play either the abusing or the abused role). Self-harm is common. These patients may also manifest a wide range of neurotic symptoms, generalised anxiety and depersonalisation being common. In psychoanalytic terms, they use the primitive defences of splitting and projection.

We have found some success in using 'sequential diagrammatic reformulation' in the cognitive analytic therapy of such patients, in particular the more difficult ones.

Cognitive analytic therapy

Cognitive analytic therapy (CAT; Ryle, 1990) is an integrated, time-limited therapy suitable for use in a public health service. It draws upon a range of psychotherapeutic theories and methods, notably cognitive behavioural approaches and the object relations school of psychoanalysis. As its central feature, it lays particular emphasis on the reformulation of patients' problems; this involves accurate description, at the most general level attainable, of what the patient does to maintain his difficulties, but does not involve the interpretation of unconscious processes. Reformulation is carried out over the first few sessions in collaboration with the patient. A written description of the patient's history is linked with brief descriptions of his current maladaptive strategies (the 'target problem procedures'). The maintenance of intentional acts or roles is seen to be controlled by mental, behavioural, and environmental factors, linked in sequence. Intentional action is organised through a hierarchy of 'procedural sequences' which is normally open to revision by new experiences; neurotic procedures are generally not open to such revision, because of 'traps', 'dilemmas', and 'snags' (Ryle, 1979). Transference is understood in similar terms.

A patient's target problem procedures (TPPs) form the basis of therapy. Patients are asked to monitor and record their current uses of their TPPs in order to help them to recognise and in due course to modify them. Cognitive behavioural treatments are also used. The key role of the therapist in CAT is to help patients to identify their TPPs and to teach them to use them.

Sequential diagrammatic reformulation

Cognitive analytic therapy has proved effective (Brockman *et al*, 1987), and has been applied in a wide range of settings. However, the brief written descriptions used are not always sufficient for more disturbed patients, and for such patients the additional use of diagrams has proved to be of value. The form of these diagrams was influenced by Horowitz (1979), who described the analysis of transcripts of psychotherapy sessions by identifying different 'states of mind' and by plotting transitions between these states, a method he called 'configurational analysis'. A state of mind (in cognitive and psychoanalytic terms) is described with reference to the typical mood, the defensive organisation, the sense of self, and the sense of other. This retrospective approach was adapted in CAT to reformulate patients' problems after four to five sessions. The agreed sequential diagramatic reformulation (SDR) is then used to guide therapeutic interventions, and as a basis for patients' self-monitoring.

The main source from which a SDR is drawn up is the clinical interview, but patients also contribute through their own self-monitoring, written tasks, and reading. The aim is to identify and characterise the patients' principal mental states, and to work out how transitions occur between them. The end result, characteristically, is a linked set of state descriptions of which one, the 'core state', represents the long-term, unresolved psychic pain of the patient. Patients may be fully or only partially or occasionally aware of this central state; characteristically they avoid it. There is often one dominant 'coping mode', such as desperately pleasing others, which has served as a source of self-esteem since childhood but which is usually accompanied or followed by shallow or unpleasant feelings. Other states may be characterised by unrewarding interpersonal strategies and some may involve somatic, behavioural, or psychological symptoms.

As the sequence of transitions between all the states is worked out, the persistence of the patient's difficulties becomes explicable, for the arrows inevitably return to the centre, showing how the core state is maintained.

Uses of SDR

SDR is of value to the clinician in that it aids accurate identification of a patient's state shifts and of the clinician's own shifting responses, enabling him to avoid collusion (see case 1 below). The map also indicates where destabilisation of the system is most likely to be effective, and is hence a guide to therapeutic intervention. Such maps could be of use to staff in in-patient or day hospitals, where patient management can often evoke 'coping-mode' conformity or provoke ill-understood acting out (see case 2 below).

For the patient, the SDR provides visible evidence of being understood and is frequently dramatically effective in containing anxiety and in initiating a co-operative attitude (see case 3 below). Patients are encouraged to use their SDRs themselves so that they need not suffer passively and feel out of control. One patient at follow-up interview quoted her therapist as having said "you may be a ship of fools but you can be the captain". She described how the daily charting of her state in this metaphorical role of captain had enabled her to maintain a markedly more stable and nearly alcohol-free existence for the first time for some years.

Case reports

Case 1: Mariette (therapist MM)

Mariette requested psychotherapy at the age of 41 following the end of her relationship with a 33-year-old man. She described uncontrollable moods, outbursts of inappropriate anger and crying, and an increased use of alcohol and cannabis. She had had a number of previous relationships with men, in all of which she became bored after a few months, at which point she would either leave or provoke rejection. She had recently ended her closest relationship yet. When not in relationships she tended to feel bored and empty.

Mariette had emerged from an insecure childhood as capable of and dependent on high achievement. She was currently a successful and ambitious magazine editor. She had had psychotherapy on two previous occasions (for two years and for six months) without feeling any lasting benefit.

She was offered 16 sessions. In the first three she was sometimes aggressive, although on occasions she became vulnerable and then accused the therapist of being insensitive and hurtful. The SDR (Fig. 1) was constructed at session 4 and thereafter she became more open and more able to work in therapy. By the end she could acknowledge that the therapy had provided the experience of being emotionally involved and vulnerable without being damaged. The diagram in this case was helpful as a guide to the therapist in dealing with the shifting aspects that Mariette presented and to the patient in providing a basis for a more coherent sense of self.

Case 2: Jacob (therapist AR)

Jacob, a 26-year-old graduate, had exhibited a schizoid pattern of emotional remoteness and restricted relationships during his adolescence and early adult life. He was assessed while a psychiatric in-patient at another hospital and was seen weekly while the attempt was made to arrange intensive in-patient psychodynamic therapy. For the past two years his schizoid withdrawal had been replaced by an unstable state with features of borderline personality disorder, namely persistent depersonalisation experiences and a mounting sense of futility and despair. Over this period he had had a number of admissions and had made five suicide

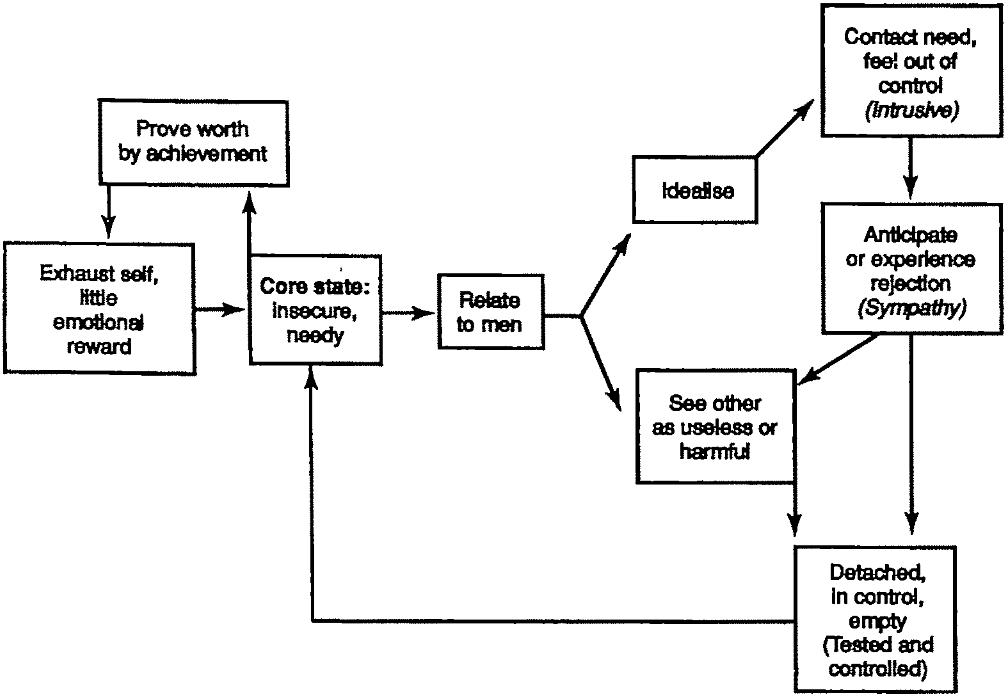
Prove worth by achievement
Exhaust self, little emotional reward
Core state: insecure, needy
Relate to men
Idealise
Contact need, feel out of control (Intrusive)
Anticipate or experience rejection (Sympathy)
See other as useless or harmful
Detached, in control, empty (Tested and controlled)

FIG. 1 Sequential diagrammatic reformulation: Manette (case 1) (counter transference in parentheses).

attempts. He was an anxious, deeply unhappy young man. Self-monitoring made it clear that his depersonalisation experiences followed the emergence of feelings of being misunderstood, accompanied briefly by considerable anger. On rare occasions the anger was vehemently expressed to the doctors and nurses looking after him, but he was usually either reserved and compliant or inert and passively resistant.

Jacob was born a year after an elder sibling who had been described as furious and uncontrolled from birth, and who had become schizophrenic at the age of 16. Jacob, by contrast, was quiet, docile, and close to his mother. He was successful at school. From his earliest years, however, he had a sense of feeling different and cut off, and he compensated for this with an elaborate fantasy life; from adolescence onwards this included homosexual fantasies of being cared for. He developed an interest in, and considerable talent for, imaginative writing. He had enjoyed his years at university but, on starting work, found the demands placed upon him increasingly meaningless and confusing; it was at this point that his despair and depersonalisation experiences began.

Jacob was seen before the development of SDR, and the diagram (Fig. 2) is a version made later by the therapist. The verbal reformulation, worked out with Jacob, identified two dilemmas: firstly that, for him, the choices seemed to be to live either in the fantasy world of care and understanding or in the painful and unmanageable real world; secondly, within the real world, the choice was seen as being either totally compliant (his historical survival mode, which he experienced as increasingly intolerable) or furious and mad (like his sibling), a role so unacceptable that he became depersonalised or suicidal. The ordinary expectations placed on him in his role as an in-patient felt to him like one more set of demands for compliance. Sadly, before his admission to a therapeutic unit could be arranged, he succeeded in killing himself.

Case 3: Pearl (therapist HB)

Pearl, aged 18, was referred to a community mental health centre by workers in a hostel where she was living. She had recently left home, against her parents' wishes. She came from a non-European Muslim background, the family having arrived in England a few years previously.

The referral followed a brief admission to a psychiatric unit from which she had discharged herself. The admission had been arranged from a casualty department where she had presented in an amnesic and almost mute state following a disturbed outburst at the hostel. Her recent behaviour had included verbal abuse, excessive drinking bouts, a preoccupation with knives, and taking an overdose of medication prescribed for a physical condition. She denied any awareness of these incidents, stating that they had occurred "in her sleep". At her initial assessment interview, Pearl reported a history of deprivation and

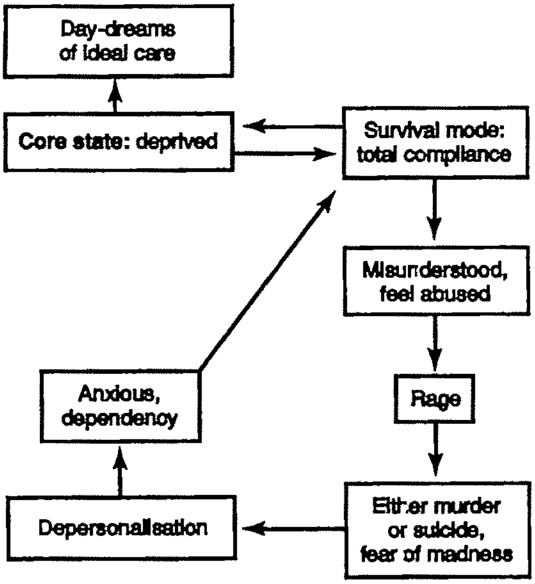
Day-dreams of ideal care
Core state: deprived
Survival mode: total compliance
Misunderstood, feel abused
Rage
Either murder or suicide, fear of madness
Depersonalisation
Anxious, dependency

FIG. 2 Sequential diagrammatic reformulation: Jacob (case 2).

severe abuse, but in doing so claimed that she was "very happy", and she denied any connection between her past experiences and her current disturbed behaviour. She was deeply ambivalent about seeking help. She was considered to meet the criteria for a diagnosis of borderline personality disorder.

Pearl was brought up initially away from her family by a couple who returned her to her natural parents when she was five. From that age until she was 14 she was sexually abused by a neighbour. It later emerged that Pearl had submitted to this continual abuse to protect her younger sister, the man saying he would otherwise do the same to her. She perceived her upbringing as having been punishing, strict, emotionally depriving, and said that she had been desperately lonely. She saw herself as a 'stranger' in the family, and felt she was treated differently. She was beaten by her parents and frequently accused of being the source of the family's ill fortunes. She gave a history of frequent overdoses and self-cutting, none of which had been severe enough to induce intervention by the family or external agencies. She had not received any previous psychological or psychiatric help. In the first two sessions Pearl described disturbing life events vividly while expressing contradictory attitudes and feelings towards them, often within a sentence or two of each other. During these sessions her state switched between excited overactivity, anger, self-hate, and distress. She feared that she might lose control and go "crazy", and she expressed strong suicidal ideas.

By the third session the therapist felt confused and exhausted. At this point she suggested that they look at these different parts of Pearl. Pearl, with apparent ease, proceeded to introduce the therapist to "crazy", "angry", "smart", and "happy" Pearl, each description being accompanied by observable changes in her body posture and vocal tone. "Crazy" and "angry" Pearl were characterised by feelings of isolation, distrust, omnipotence, and fear, and by destructive acts, seen as the result of powerful parts of herself which assumed control against her will. "Smart" Pearl was characterised by conformity, placation, and careful control. "Happy" Pearl had no past, was overactive, playful, exuberant, trusting, and caring; this was Pearl's preferred state and was referred to by her in the first person. The therapist remarked to Pearl that she had also seen a "sad" Pearl, who was then acknowledged briefly as being hurt, lonely, in need of help, and wanting happiness. She stated that "angry" and "crazy" Pearl were furious that

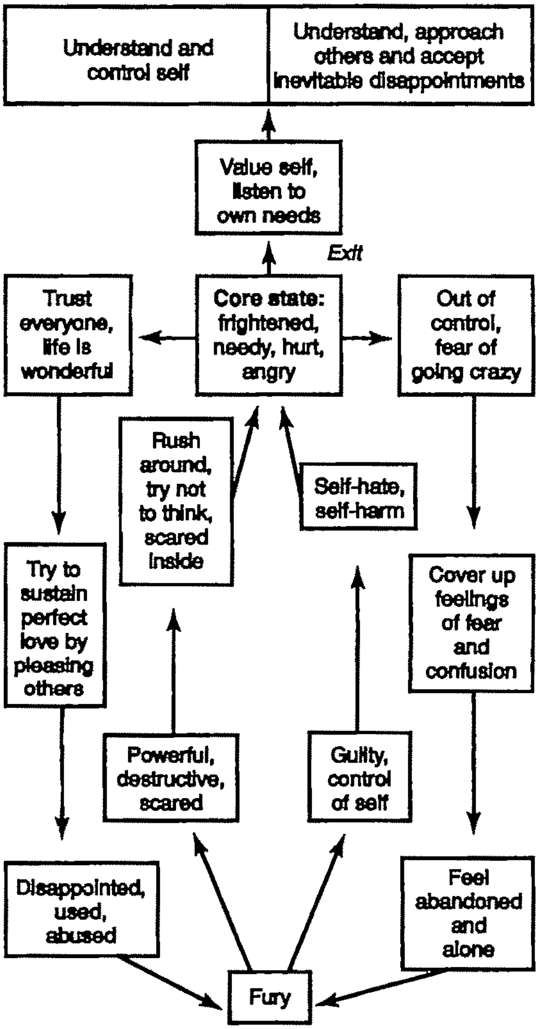
Understand and control self
Understand, approach others and accept inevitable disappointments
Value self, listen to own needs
Exit
Trust everyone, life is wonderful
Core state: frightened, needy, hurt, angry
Out of control, fear of going crazy
Rush around, try not to think, scared inside
Self-hate, self-harm
Try to sustain perfect love by pleasing others
Cover up feelings of fear and confusion
Powerful, destructive, scared
Guilty, control of self
Disappointed, used, abused
Feel abandoned and alone
Fury

FIG. 3 Sequential diagrammatic reformulation: Pearl (case 3).

their presence had been revealed but that "happy" Pearl was very pleased. The possibility of these different parts becoming more familiar with each other, in order to help her feel more in control of her life, was proposed.

At the next session, the attempt was made to bring all of Pearl's parts into the room together, linking them to her core state of deprivation and suffering. Pearl connected some of her current feelings to her past abuse, and the rest of the session became an abreactive release of some of the pain she had held onto for so long. She not not keep her next appointment but responded to a letter. During this time a worker from the hostel reported concern about Pearl's self-destructive behaviour, for which she was at risk of being asked to leave.

In the ensuing session, the therapist worked with Pearl at the task of identifying the sequence of transitions between Pearl's states, and at the sixth session the SDR was finalised (Fig. 3). Pearl had worked with the therapist at the task of describing her states and state transitions, and by the time the reformulation task was completed at the sixth session, she reported that she felt that she had already changed. She took up the 'exit' suggested in the SDR, namely approaching others, and using her written reformulation and SDR to explain to her peer group and to the hostel staff her recent behaviour and her needs. Her improved self-esteem and behaviour were confirmed by the hostel staff.

Conclusion

Ross & Gahan (1988), in reviewing the treatment of patients with multiple personality disorder, reported that successful treatment required the explicit goal of personality integration and the use of diagrammatic reformulation. Our own work with less dissociated personality disorders suggests a more extensive use of this approach. The aim of psychotherapy or of day hospital or in-patient care of patients with disordered personalities is to aid their self-control and integration. Those in contact with these patients must be able to resist the strong pressures to collude with their pathological processes if this aim is to be achieved. We believe that a jointly produced SDR can make an important contribution to management and therapy, and can assist patients in their own self-understanding and control.

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Who Knows Best? Family Interaction and Eating Disorders

GLENN WALLER, PETER SLADE and RACHEL CALAM

Links have been established between eating disorders and abnormal patterns of interaction between members of the sufferer's family. Faced with often contradictory information, the clinician needs to know which member of the family will give the most valid description of that family's interaction. This study demonstrated the value of the Family Assessment Device (FAD) as a measure of 'pathology' in the families of anorectic and bulimic women. Using the ability to predict the eating disorder as an index of concurrent validity, the sufferers themselves had the most realistic perceptions of their families' interactional styles.

Clinicians have long held that family interaction might be linked to anorexia nervosa and bulimia nervosa (e.g. Palazzoli, 1974; Minuchin *et al*, 1978), and research has tended to support that hypothesis (Kog & Vandereycken, 1985). There have been several studies of how family styles relate to both eating attitudes (Kagan & Squires, 1985) and eating disorders (Heron & Leheup, 1984).

The chief difficulty with drawing general conclusions from this research is that it has used a wide variety of assessment methods, thus making the results difficult to compare. In order to relate family interaction to abnormal eating, it would be valuable to have a common model of family functioning. Previous research has shown that a promising candidate is the McMaster model of family functioning (Epstein *et al*, 1978). This model comprises six dimensions of family interaction: the family's style of resolving problems (problem solving); clarity of information transmission (communication); differentiation of tasks (roles); experience of emotions (affective responsiveness); concern for each other (affective involvement); and clarity of rules (behaviour control).

The McMaster model fulfils several important criteria for use as a tool in clinical work and in research. First, it is easy to administer, using the Family Assessment Device (FAD) in a questionnaire or a structured interview form (Epstein *et al*, 1983; Miller *et al*, 1985). These are contemporary rather than retrospective measures of family interaction. Second, the model differentiates normal families from those with members who suffer from particular clinical disorders. For example, the FAD shows abnormal perceived interaction in the families of patients with major depression, but not in those with bipolar disorders (Miller *et al*, 1986). Finally, the McMaster model suggests developmental tasks for the family (Will & Wrate, 1985), and thus aids the clinician in planning and evaluating treatment.

Miller *et al* (1985) described the psychometric properties of the FAD as acceptable. In particular, it had good internal consistency, although its test–retest reliability was less satisfactory. However, use of the McMaster model in assessment (via the FAD) has been largely confined to the Butler/Brown team that developed it (Epstein *et al*, 1983) in the USA. The FAD has not been widely used in other cultures, suggesting that its value has still to be established where different cultural norms apply (e.g. Lau, 1984). Overall, the FAD seems to be a useful tool, although attention needs to be given to its cultural and psychometric assumptions in interpreting it for clinical use.

Even armed with a useful tool for evaluating families, difficulties remain for the clinician. In particular, which member of the family will give the best description of the family's functioning? Those who work with families where a member has an eating disorder will be used to receiving conflicting accounts from different family members. Blame will often be attributed externally, in order to deny personal responsibility for the cause or the relief of the eating disorder. However, it is important to know whose account has the greatest clinical validity. In this case, the most meaningful measure of concurrent validity is the existence of a diagnosable eating disorder.

This study compares anorectic and bulimic women's perceptions of family interaction with their parents', using the questionnaire form of the FAD. In particular, it focuses on the issue of whose perceptions of family interaction are best at predicting the existence of eating disorders.

Method

Subjects

Forty-eight 'clinical' and 30 'comparison' families participated. Each family consisted of at least three members: daughter, mother, and father. In each clinical family, the daughter was suffering from an on-going, diagnosable eating disorder. No male anorectics or bulimics were referred during the course of the study.

Diagnoses according to DSM-III-R (American Psychiatric Association, 1987) criteria were made after clinical interview. Anorexia nervosa (category 307.10) was defined as a disorder involving low body weight for height (at least 15% below normal); fear of weight gain; disturbed body image; and absence of at least three consecutive menstrual cycles. Bulimia nervosa (category 307.51) was defined by recurrent episodes of binge-eating (averaging two or more episodes per week over the previous three months); perceived lack of control during these episodes; use of vomiting, purging, diuretics, fasting, or exercise to prevent weight gain; and overconcern with body shape or weight. Fourteen daughters were classified as suffering from anorexia nervosa, and 34 from bulimia nervosa. Two volunteering families were not used, on the grounds that their daughters failed to meet the DSM-III-R weight-loss criteria for their diagnosis (anorexia nervosa), although they met Russell's (1970) criteria. None of the daughters in the comparison families reported being diagnosed as having any psychiatric disorder.

The clinical families were recruited on a voluntary basis from clinical practices and self-help groups in the north-west of England. None were involved in family-orientated therapies. Initially the sufferers were approached, and asked to pass on questionnaires to their parents. The 30 comparison families were recruited via their daughters, who worked or studied in a variety of settings in the Liverpool and Manchester areas. The daughter and mother participated in all 78 cases. However, only 39 fathers returned the questionnaire.

Assessment instruments

The FAD self-rating questionnaire (Epstein *et al*, 1983) was used for all families. This questionnaire consists of 53 items on seven scales. Six of these scales (problem solving, communication, roles, affective responsiveness, affective involvement, behaviour control) address the dimensions of family functioning suggested by the McMaster model. The remaining scale is a measure of general functioning. The family member rates his or her family on a four-point scale for each item, and mean scores for each scale are obtained between 1 (extremely 'healthy') and 4 (extremely 'unhealthy').

The daughter and her parents were asked to complete the FAD (separately from each other), giving their perceptions of the whole family. They then returned their questionnaires in person or using a reply-paid envelope.

Results

Table I shows the physical characteristics of the daughters in each group of families. The anorectics were significantly lighter for their height (as assessed by the Body Mass Index (BMI) of Llewellyn-Jones & Abraham (1984)) than the

TABLE I
Means (s.d.) for daughters' physical characteristics and ratings of family functioning

	Comparison group (n=30)	*Anorexia nervosa (n=14)*	*Bulimia nervosa (n=34)*	F
Age: years	26.3	26.3	25.1	0.21
	(8.25)	(6.46)	(7.90)	
Height: cm	161.5	160.0	160.3	0.37
	(5.51)	(6.08)	(8.22)	
Weight: kg	58.9	40.9	52.7	18.9 **
	(9.81)	(4.62)	(9.69)	
Body Mass Index	22.5	16.0	20.5	22.0 **
	(3.11)	(2.02)	(3.29)	
FAD scale				
Problem solving	2.00	2.54	2.74	10.9 **
	(0.46)	(0.48)	(0.81)	
Communication	2.20	2.65	2.60	5.00*
	(0.56)	(0.40)	(0.65)	
Roles	2.04	2.36	2.56	10.9 **
	(0.37)	(0.46)	(0.52)	
Affective responsiveness	2.07	2.53	2.65	7.16*
	(0.71)	(0.54)	(0.67)	
Affective involvement	2.29	2.53	2.57	8.85**
	(0.21)	(0.30)	(0.32)	
Behaviour control	1.68	2.16	2.04	14.9 **
	(0.22)	(0.31)	(0.40)	

*$P<0.01$; **$P<0.001$.

bulimics, who in turn were lighter than the comparison women. However, there were no differences in the mean ages of the groups. Nor was there any evidence of a difference between the groups in social class.

Ratings of family interaction

Tables I and II show the mean ratings by the family members on the six scales of interaction. They also identify the differences between the groups, using analyses of variance.

The daughters' ratings (Table I) did not distinguish anorectics from bulimics, but differentiated the clinical groups from the comparison women on all six scales (Tukey's HSD (Kirk, 1968), $P<0.05$ in all cases). The anorectic and bulimic women also perceived their families as less 'healthy' than the comparison group in all cases.

In contrast, mothers (Table II) of anorectic and bulimic women rated their families as less 'healthy' than comparison women did on only two scales (affective responsiveness and affective involvement), although their ratings also failed to differentiate the clinical groups. The fathers' ratings (Table II) failed to differentiate the clinical and comparison groups on any scales. The smaller number of fathers who opted to participate may have been relevant to this outcome, although the small F values suggest otherwise.

Thus, there were differences in the patterns of family members' perceptions of their interaction, and in the abilities of those perceptions to differentiate clinical and comparison families. However, the FAD did not distinguish the anorectic from the bulimic women, suggesting that they may have similar perceived family pathologies. Which family member gives the most useful picture of family interaction, in terms of predicting the daughter's diagnostic status?

Concurrent validity

Discriminant function analysis was used to predict the presence or absence of eating disorders from the six scales of the FAD. Table III shows the classification results of the separate analyses for the daughters', mothers', and fathers' ratings. As there were no differences between the anorectic and bulimic women on the FAD, they were combined into a single clinical group.

For the daughters, only affective involvement, affective responsiveness, problem solving, and behaviour control were necessary to form the discriminant function that

TABLE II

Means (s.d.) of parents' ratings of family functioning

	Comparison group	*Anorexia nervosa*	*Bulimia nervosa*	F
Mothers				
No.	30	14	34	–
Problem solving	1.95 (0.34)	2.18 (0.43)	2.14 (0.58)	1.63
Communication	2.18 (0.34)	2.18 (0.50)	2.37 (0.42)	1.80
Roles	2.35 (0.45)	2.41 (0.49)	2.34 (0.47)	0.13
Affective responsiveness	1.87 (0.60)	2.48 (0.48)	2.37 (0.73)	6.29*
Affective involvement	2.29 (0.26)	2.56 (0.35)	2.59 (0.37)	7.04*
Behaviour control	1.77 (0.32)	1.93 (0.31)	1.88 (0.38)	1.42
Fathers				
No.	21	6	12	–
Problem solving	1.88 (0.44)	1.83 (0.29)	2.06 (0.35)	0.91
Communication	2.18 (0.43)	2.38 (0.29)	2.28 (0.37)	0.63
Roles	2.28 (0.50)	2.53 (0.56)	2.36 (0.47)	0.60
Affective responsiveness	2.08 (0.74)	2.37 (0.52)	2.28 (0.47)	0.60
Affective involvement	2.30 (0.24)	2.46 (0.28)	2.40 (0.30)	1.01
Behaviour control	1.81 (0.38)	1.81 (0.35)	1.71 (0.37)	0.31

*$P<0.01$.

TABLE III
Prediction of eating disorders from ratings of family interaction by individual family members

Actual group	*No. of cases*	*Predicted group* *Comparison*	 *Clinical*
Daughters' ratings			
Comparison	30	24	6
Clinical	48	5	43
		Correctly classified 85.9%	
Mothers' ratings			
Comparison	30	20	10
Clinical	48	6	42
		Correctly classified 79.5%	
Fathers' ratings			
Comparison	21	15	6
Clinical	18	7	11
		Correctly classified 66.7%	

significantly differentiated the clinical and comparison groups (*F* (4,73) = 14.29; *P* < 0.001). This agglomerated function correctly categorised 85.9% of clinical and comparison families (against a baseline of 38.5% if it had simply assumed all of the families to be 'normal'), so the rate of successful identification was high. However, there were several false positives. Twenty per cent of comparison women were classified as belonging to the eating-disordered group, which is similar to or lower than the false positive rate reported by Miller *et al* (1985), who used individual scales of the FAD rather than an agglomeration.

The mothers' ratings of affective responsiveness, affective involvement, and roles formed a function that differentiated the groups (*F* (3,74) = 10.0; *P* < 0.001). This function was less accurate (79.5% correct) than the daughters' ratings, but still much higher than the baseline (of 38.5%). Most noticeably, they had a higher rate of false positives (33.3%) than their daughters.

The fathers' ratings were much poorer than the other family members' in categorising the groups. Their discriminant function did not differentiate the groups (*F* (3,35) = 1.64; *P* = 0.20). Their perception of family interaction was poor at classifying the clinical and comparison families (66.7% correct, against a baseline of 53.8%).

Thus, the daughters' ratings of family interaction had the greatest concurrent validity (predicting their eating disorders). The mothers' and fathers' ratings were less valid and less useful, though to different degrees. However, all family members made a number of errors in classification compared with the diagnostic procedure.

Discussion

The three family members' perceptions of family interaction differed widely, as did their abilities to predict the presence or absence of eating disorders in the daughter. The daughters' own ratings of family interaction were the best predictors of whether or not they could be diagnosed as suffering from an anorectic or bulimic eating disorder. Mothers were fairly accurate, but their rate of false positives was higher than that of their daughters – a major difficulty when dealing with a relatively uncommon disorder in a large population. Fathers' ratings had virtually no predictive power, suggesting that they did not share their daughters' perceptions of the family interaction as problematic.

The immediate clinical relevance of these findings is that the clinician who wishes to understand how perceived family interaction is related to eating disorders should attend to the anorectic or bulimic sufferer's experiences of her family. Her parents' perceptions will be less immediately relevant. However, it may be necessary to make parents aware of their daughter's experience. Therefore, their perspective will be of some value.

Given the trade-off between sensitivity and specificity of the FAD, it cannot serve in isolation as a clinical tool. It is probably of greatest value in conjunction with diagnostic interviews, giving an understanding of family issues in individual cases. The classification errors of the FAD are common to measures of family function when attempting to differentiate clinical and comparison families. They occur in both questionnaire ratings (e.g. Olson & McCubbin, 1983) and observational measures, such as expressed emotion (e.g. Vaughn & Leff, 1976). Miller *et al* (1986) concluded that these measures have similar rates of misclassification to the FAD, and suggested some potential explanations for this common theme.

Daughters' ratings of their families' affective involvement and responsiveness, problem solving, and behaviour control were powerful predictors of eating disorders. These conclusions are related to those of other clinicians and researchers. For example, Pole *et al* (1988) have shown that parental care and protection are related to bulimic disorders. Similarly, Minuchin *et al* (1978) described the roles of family cohesion and flexibility. Extrapolating from the different sources, abnormal emotional links and strict behaviour control within families appear to be strongly related to eating disorders.

Different conceptual models of family pathology have been proposed, and each has a different value for the clinician. An important issue in deciding the value of these models is that they tend to have the common theme of giving multidimensional descriptions of the family. The number and nature of these dimensions vary, although Miller *et al* (1985) have described how the models may be conceptually and practically inter-related. Use of an alternative measure in the present research might have yielded fewer dimensions of perceived pathology, and might therefore have made for a simpler description of family functioning. However, the FAD has the benefit of describing dimensions of interaction that can be conceptualised easily for treatment purposes (Will & Wrate, 1985).

The clinician with an interest in family-orientated therapies will need to consider helping the family to set and express appropriate levels of concern for each other, and to establish clear, workable rules about behaviour and dealing with problems. Will & Wrate (1985) have given more complete schemes of the goals involved for families when their interaction is rated as 'unhealthy' in these ways. Alternatively, a clinician working within a psychodynamic or cognitive framework will need to examine the factors that have led to the sufferer's perception of his or her family. Further clinical research is needed to test the efficacy of different forms of therapy in such cases. Russell *et al* (1987) have begun to demonstrate the conditions under which certain therapies are more effective in the eating disorders. The present findings suggest that emotional expression and communication about rules might be valuable targets of interventions, or even simple means of evaluating treatment, regardless of the therapeutic model employed.

Further research would be useful in two areas. First, the direction of causality could be investigated. A prospective study would help to establish whether family interaction leads to eating disorders, or vice versa. The present study shows that the FAD would be a valuable tool in such research, and that the daughters' own perceptions should be the prime focus. Second, it is known that the pattern of family interaction differs for particular disorders, so how might this specific style of interaction explain the development of eating disorders? Slade (1982) suggested that family interaction may be related to the low self-esteem and perfectionism that 'trigger' eating disorders. Again, prospective research would help to expand on this link, and the FAD would be a meaningful measure of family interaction.

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Time Course of Cerebral Magnetic Resonance Changes after Electroconvulsive Therapy

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Nuclear magnetic resonance images of the non-dominant cerebral hemisphere were obtained in 20 unipolar depressed patients immediately before and 25 minutes after electroconvulsive therapy (ECT). T_1 values rose about 1%. Repeated scanning up to 24 hours after ECT was carried out in 13 of these patients. The greatest change in magnetic resonance images was two hours after ECT, and thereafter images gradually returned to baseline values. There was no correlation between magnetic resonance changes and the time taken to become re-orientated after ECT.

Magnetic resonance imaging (MRI) does not use ionising radiation and thus it is possible to scan patients repeatedly without hazard. Mander *et al* (1987) studied the effects of electroconvulsive therapy (ECT) on brain images produced by MRI. The T_1 relaxation time (a reflection of brain water content) rose within 30 minutes of ECT, but there was no cumulative increase in T_1 values over a course of treatment. In three patients the maximum increase in T_1 occurred four to six hours after ECT. These workers suggested that the increase in T_1 was a consequence of a temporary breakdown in the blood–brain barrier.

This finding may be of importance to theories of the mode of action of ECT. Fink (1979) has hypothesised that the mode of action of ECT depends on a breakdown in the blood–brain barrier that permits the entry of behaviourally active peptides which would not otherwise reach the central nervous system. This is the so-called 'neuroendocrine hypothesis' of ECT. These findings may also be relevant to the adverse cognitive effects of ECT (Weiner, 1984).

The major aim of this study was to attempt to replicate the findings of Mander *et al* (1987) and to study the effects of ECT on the T_2 relaxation time (spin–spin relaxation), which was not examined in the previous study. A second aim was to investigate any possible relationship between MRI changes and the adverse cognitive effects of ECT.

Method

Pilot volunteer study

One aim of the study was to make T_1 measurements over several hours after ECT. In order to study the reliability of T_1 measurements, five volunteers (3 men, 2 women, mean age 30 years) were scanned at three-hourly intervals on different days.

Patient study

Twenty depressed patients were scanned immediately before and 25–30 minutes after ECT. Thirteen of them were scanned immediately before treatment and up to five times after treatment (+25 minutes, +2 hours, +4 hours, +6 hours, +24 hours). All patients undertook tests of orientation before and after ECT, and 18 patients undertook a logical reasoning test immediately before and throughout the day of ECT. Patients fast before ECT, which may affect magnetic resonance images; thus, osmolality was measured immediately before and six hours after ECT.

Patients were studied only when their depression had started to lift and the clinical team felt they were able to take part in the study. Patients were studied on average at ECT number 4.6 (second to seventh treatment)

All patients satisfied Research Diagnostic Criteria (RDC; Spitzer *et al*, 1978) for major depressive disorder. The sample consisted of five men and 15 women of mean age 53 years (range 27–78 years). None had a history of mania, head injury, brain damage, or alcohol abuse, and none had received ECT in the previous three months.

The anaesthetic for ECT was standard. Methohexitone was used to induce anaesthesia (70–100 mg) and suxamethonium was used to induce muscle relaxation (25–60 mg). Right unilateral ECT ($n = 8$) and left unilateral ECT ($n = 1$) were administered with electrodes in D'Elia's position, and bilateral ECT using the bitemporal position. The electrical stimulus was standard and provided from a MECTA machine (800 mA, 70 Hz, 1.5 ms, 2 s). Seizure duration was estimated by observation only and tonic–clonic activity timed by a stopwatch. Mean seizure duration was 30 seconds (5–48 seconds).

Measurement of T_1 and T_2

An M&D Technology Ltd 0.08 tesla resistive magnetic resonance imaging system was used. The T_1 and T_2 measurements were made on 12-mm-thick transverse sections of the brain 10 mm above the widest point of the lateral ventricles. Measurements of mean T_1 and T_2 of the entire area of the transverse section (the grey and white matter together but excluding the cerebrospinal fluid (CSF)) were made. Using a specially designed software package, a cursor was used to outline the non-dominant hemisphere (the boundary of the area to be measured) directly on the image of the transverse section as it was displayed on a monitor. CSF has a T_1 value much higher than either white or grey matter, and is brightly coloured on the monitor display. The outline of the cerebral hemisphere was made in such a way as to exclude CSF. Each scan was measured independently by two raters (AIFS and RHBD), and the results below are averages of these two measurements. The secretary in the NMR Unit gave each rater a list of the code numbers of scans to be measured. The timing of scans for each patient was disguised by jumbling the order of the code numbers. The code number of a scan is displayed on the monitor when T_1 and T_2 measurements are made. This code number can theoretically reveal the time of the scan by reference to a log-book kept in the Unit. However, this was never done by either of the raters at the time of measurement.

Cognitive measures

Two aspects of cognition were assessed before and after ECT. First, orientation was assessed about 30 minutes before ECT, and the time to become re-orientated after treatment was measured in minutes. The tests of orientation were patient's name and age, day and date, name of hospital, and principal researcher's name. The second measure was a two-minute reasoning test of proven reliability (Baddeley, 1968). The test was adapted to be used on a microcomputer. The patient had to study a video screen on which the letters AB or BA appeared. A statement appeared below (e.g. "A follows B") and the patient had to decide whether this statement was true or false and press either T or F on the keyboard. Each patient was allowed to practise the test on the afternoon before ECT and the morning of ECT. The reasoning test was carried out after each scan.

Statistics

The agreement between the two raters in measuring T_1 and T_2 was assessed by Pearson product-moment correlation. The changes in T_1 and T_2 after ECT were assessed by paired *t*-tests. There was an *a priori* hypothesis derived from the findings of Mander *et al* (1987) that T_1 would rise after ECT and thus one-tailed *P* values were used for T_1, but two-tailed *P* values were used for T_2 and elsewhere. The correlation between NMR changes and clinical variables was assessed by either Pearson product-moment correlation or Kendall's method, depending on the distribution of the data.

Results

The product-moment correlation between the two raters for T_1 was $r=0.87$ ($P<0.001$), and for T_2 was $r=0.90$ ($P<0.001$).

The T_1 measurements in the five volunteer subjects who were scanned at three hourly intervals on different days suggested that T_1 measurements were consistent throughout the day (9 a.m. $T_1=331.2\pm3.5$, midday $T_1=330.4\pm3.2$, 3 p.m. $T_1=331.6\pm3.6$).

Table I shows that T_1 rose on average by 3.3 ms 25 minutes after ECT. There was a smaller, non-significant rise in T_2. There were no statistically significant differences between unilateral and bilateral ECT in the effects on T_1 or T_2, nor were there any significant right–left differences in patients receiving unilateral ECT.

TABLE I

T_1 and $T_2\pm$s.e. (ms) of non-dominant hemisphere before and 25–30 minutes after ECT (n = 20)

	Before ECT	*After ECT*
T_1	338.3 ± 2.9	341.6 ± 2.7*
T_2	105.1 ± 1.1	106.1 ± 1.2

*$P<0.05$.

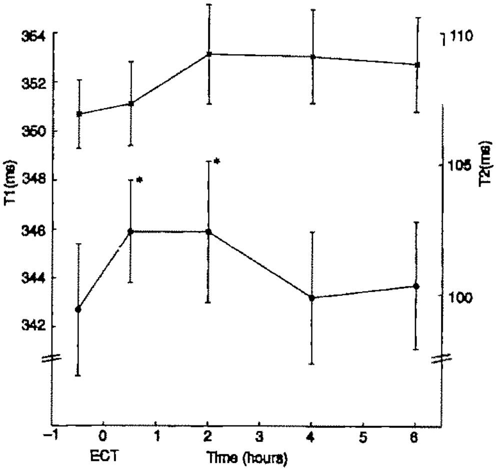

FIG. 1 $T_1\pm$s.e. ($n=13$) (○——○) and $T_2\pm$s.e. ($n=10$) (●——●) of non-dominant cerebral hemisphere over the six hours after ECT. *Significantly ($P<0.05$) greater than baseline value

Figure 1 shows T_1 and T_2 in the non-dominant hemisphere over the six hours after ECT. The maximum rise in T_1 occurred 25 minutes (+3.2 ms) and two hours (+3.2 ms) after ECT. By six hours after ECT, T_1 values had almost returned to pre-treatment levels, and by 24 hours they were identical to pre-treatment levels ($n=10$). The maximum rise in T_2 was two hours after ECT (+2.4 ms), but this rise was not statistically significant. By six hours after ECT, T_2 was still slightly raised, but had returned to pre-treatment values 24 hours after ECT ($n=9$).

The times to become re-orientated for each of the orientation questions were totalled. The time to re-orientation was far greater in those patients who received bilateral ECT than in those who received unilateral ECT (322.8 ± 67.3 minutes v. 76.8 ± 14.9 minutes, $P<0.01$). Surprisingly, the performances on the reasoning test steadily improved after ECT in both sets of patients. The likeliest explanation is that this was a practice effect which happened despite the practice given the day before and on the day of treatment. However, there is also a diurnal rhythm in cognition, and performance efficiency is normally lowest in the early morning (Folkard, 1986). In addition depressed mood may have lifted after ECT, but this was not assessed. Forty minutes after treatment the improvement in performance among those patients treated with unilateral ECT was

10.2 ± 6.2 but only 1.4 ± 1.6 in those treated by bilateral ECT (not statistically significant).

There were no statistically significant correlations between NMR changes 25–30 minutes after ECT and age, weight, dose of anaesthetic drugs, treatment number, fit length, osmolality, or any of the cognitive changes after ECT. The maximum changes in T_1 and T_2 were seen at two hours after treatment. The only significant finding was that age was positively correlated with the rise in T_1 (tau = 0.48, $P < 0.05$) and the rise in T_2 (tau = 0.74, $P < 0.01$) two hours after ECT.

Discussion

This study confirms the findings of Mander *et al* (1987) that T_1 relaxation time of the cerebral hemisphere rises after ECT. The rise occurs within minutes of the treatment, but T_1 values return to their original level within six hours of ECT. There was also a non-significant rise in T_2 relaxation time. The adverse cognitive effects of ECT were assessed by time to become re-orientated and the ability to perform a logical reasoning test, and were not correlated with the extent of the NMR changes.

One technical problem is the inclusion of CSF in the images of the cerebral hemisphere in which we made the T_1 and T_2 measurements. We traced the non-dominant hemisphere trying to exclude CSF, but it is likely that small volumes of CSF were included at the boundary of the area we traced (the 'partial-volume effect'). Although the present findings are similar to those in an earlier study, we cannot disprove the argument that the rise in T_1 was because more CSF (which has a very high T_1 value) was included in our post-ECT scans. This problem will become less significant when MRI instruments of greater resolution are available.

The interpretation of these findings is difficult, as many factors may influence T_1 and T_2 relaxation times (Mathur-De Vre, 1984). Anaesthesia alone may reduce T_1 values. Mander *et al* (1987) reported a non-significant fall in T_1 30 minutes after anaesthesia in five volunteers. Whitfield & Douglas (1989) also found a non-significant fall in T_1 and a significant rise in T_2 in a series of ten women two to six hours after anaesthesia for a cervical cone biopsy. Thus, the increase in T_1 may be an underestimate of the change that occurs with ECT itself and the non-significant increase in T_2 may be an effect of anaesthesia alone.

The passage of electric current through the brain induces cerebral seizure activity, and this is accompanied by a major increase in blood pressure and in cerebral blood flow. The effects of electrical current and seizure activity on cerebral magnetic resonance images are unknown. Bolwig (1984) has shown that hyperperfusion of cerebral capillaries, and not seizure activity itself, leads to increased permeability of the blood–brain barrier.

Although these findings are compatible with the neuroendocrine hypothesis of the mode of action of ECT, the change we found was small and its significance is yet to be established. The relationship between the changes on MRI and clinical improvement after ECT was not assessed. Rangel-Guerra *et al* (1982) have reported an increase in T_1 in the illness phase in bipolar affective disorder. We found no relation between these changes on MRI and the adverse cognitive effects of ECT, although only orientation and logical performance were tested. Observation alone is a crude measure of the duration of seizure activity and there may be a relation between more sophisticated measures of seizure activity and changes on MRI. The MRI changes may be a consequence of the increase in blood pressure and cerebral blood flow that occur during ECT, but the effects of anaesthesia and unipolar depressive illness on magnetic resonance images also merit further study.

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Acute Psychotic Reactions and Stress Response Syndromes Following Intramuscular Aqueous Procaine Penicillin

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Acute non-anaphylactic reactions to procaine penicillin include fear of death, sensations of disintegration, perceived changes of body shape, and visual and auditory hallucinations. Fragments of the original reaction experience, or elaborations of it, feature prominently in a chronic syndrome which persists if the initial reaction is not supported emotionally or professionally. The cultural acceptance of the reality of 'mysterious' forces played a role in the syndrome. Compatible diagnostic labels were panic disorder, post-traumatic disorder, mixed phobic disorder, and depressive illness. The observed period prevalence rate is 5.9 per 1000.

Acute non-anaphylactic reaction with psychological features, rather than the physiological shock features of true anaphylaxis, after intramuscular administration of aqueous procaine penicillin (PP) was first described by Batchelor *et al* (1951), but later designated as Hoigne's syndrome after Hoigne who reported several cases (Hoigne & Schoch, 1959; Hoigne, 1962). Tompsett (1967) listed five additional reports (Sohval, 1953; Grounds, 1960; Bjornberg & Selstam, 1960; Dennig & Schwoerer, 1963; Utley *et al*, 1966). The literature then contained 83 cases. Descriptive words like 'unusual' (Batchelor *et al*, 1951), 'severe immediate' (Sohval, 1953), 'pseudo-anaphylactic' (Tompsett, 1967), 'acute non-allergic' (Menke & Pepplinkhuizen, 1974) were used to emphasise whatever the observers thought most significant. Apart from the report of Sohval (1953), from North America, most other early reports were from the UK and continental Europe. There were, however, later reports from USA (Tompsett, 1967; Downham & Ramos, 1973; Galpin *et al*, 1974). Most reports are from clinics for sexually transmitted diseases and dermatology clinics, with the important exception of Tompsett (1967), who presented his series from a cardiology clinic, and one from a dental practice (Eggleston, 1980).

The reactions seem to be more frequent when large doses of PP are used (Green *et al*, 1979) or when inadvertently given by intravenous infusion (Galpin *et al*, 1974). There is no report from developing countries despite the fact that PP is much used and is now listed as a basic drug in many primary health care formularies. At an estimated prevalence rate of 3 per 1000 (Batchelor *et al*, 1951) there must be a large number of unreported or misdiagnosed reactions, but there is, to the best of our knowledge, only one report in the psychiatric literature (Bjornberg & Selstam, 1960). The reaction profile most commonly presented in the literature may be summarised as follows:

(a) reaction within one minute of injection (Batchlor *et al*, 1951) or before injection is completed (Tompsett, 1967, case 4)
(b) characteristic symptoms of:
  (i) extreme apprehension or fear of death (Menke & Pepplinkhuizen, 1974);
  (ii) auditory/vestibular, visual, gustatory (penicillin taste), olfactory and kinaesthetic disturbances, including illusions and hallucinations
  (iii) vasomotor disturbances such as tachycardia, systolic hypertension and, infrequently, cyanosis (observed by all authors except Batchelor *et al* (1951))
  (iv) depersonalisation, perceived changes in body weight and shape
  (v) occasional epileptiform seizures (Downham & Ramos, 1973)
(c) absence of anaphylactic features; patients often able to continue the PP schedule (32 out of 83 cases – Tompsett (1967)), and disturbances subsiding rapidly with reassurance (Batchelor *et al*, 1951) or medication such as phenobarbitone or diazepam (Downham & Ramos, 1973).

Following the suggestion of Batchelor *et al* (1951), it has been assumed that inadvertent intravenous injection of PP during intramuscular administration occurred (Galpin *et al*, 1974) allows PP (or its components) to reach the brain. This explanation has been difficult to sustain in the absence of the evidence of cardiac septal defects that would allow a by-pass of the cardiopulmonary system. It is accepted that most of the effects may be produced by procaine (Green *et al*, 1974) and by pharmacologically similar local anaesthetics (Saraway *et al*, 1987). Blood procaine levels compatible with clinical effects have been reported (Green *et al*, 1974), and alternative ways in which free procaine could reach the blood have been explored (Bell *et al*, 1954; Green *et al*, 1974).

Regarding the neurological pathway, the consensus appears to be that features observed are compatible with a limbic disorder (Menke & Pepplinkhuizen, 1974; Pollock, 1987) rather than a 'medullary storm' (Batchelor *et al*, 1951). Menke & Pepplinkhuizen (1974) have, in a retrospective analysis, identified "various psychiatric syndromes with one common denominator – the hyperaesthetic syndrome" (i.e. a pre-morbid unstable limbic state. They have compared the observed reaction profile with "phobic anxiety depersonalisation or calamity syndrome". This syndrome bears a resemblance to post-traumatic stress disorder (PTSD) in DSM-III (American Psychiatric Association, 1980). Also, because procaine can cause limbic kindling (Pollock, 1985), and as it is given with a neuroactive agent (penicillin) (Gutnick & Prince, 1975), the possibility of neurological kindling with repeated and prolonged injections of PP has been suggested (Ilechukwu, 1987).

This paper reports a chronic syndrome emerging from acute PP reactions.

Method

After an informal pilot survey, a case register was begun in our department for all new patients presenting with symptoms attributed to a reaction to PP. Patients suffering PP reactions referred by other centres as well as patients spontaneously describing such reactions during interviews were entered into the register. The total number of new cases of all disorders seen in the department over the survey period was obtained from the medical records department.

Ten physicians who had practised medicine or a medical specialty for 15 years or more were chosen and personally interviewed. They were simply asked whether they had witnessed or had been referred patients who had had any gross psychiatric symptoms following an injection of PP. Five recalled at least one case of such a reaction, three of whom were internal physicians who claimed to have witnessed such reactions following 'normal' doses (i.e. 0.8 MU) of PP.

Results

There was a five-year period prevalence rate of 5.9 per 1000 of all new patients who presented to the Department of Psychiatry, Lagos University Teaching Hospital (Table I).

TABLE I

Prevalence of PP reactions in patients at the Department of Psychiatry, Lagos University Teaching Hospital

Year	*No. of patients reacting to PP*	*All new cases*
1982	1	208
1983	1	186
1984	1	276
1985	2	226
1986	2	288
Total (1982–86)	7	1184

Five-year prevalence 5.9 per 1000.

Case reports

Case 1

Mr A, a 23-year-old university student, reached our unit ten days after he had an unusual reaction to the first dose of a course of PP. He had received PP on previous occasions without ill effects. He recalled that immediately after the injection, he was struck by an intense lightning-like flash, heard his own voice echoing as if from distant loudspeakers, and felt dizzy, weightless, chest and arms "expanding internally", and profound weakness. He had not had such an experience, nor any other psychiatric symptoms, before. He was treated as if for anaphylactic shock and admitted briefly; PP was discontinued. The symptoms recurred the next day. Following persistent complaints of being unwell, he was given chloroquine injection with promethazine. This was also followed by a reaction almost as intense as the first. A physician relative later insisted on hospital treatment.

At our clinic he continued to complain about bright lights, a feeling of emptiness which persisted in spite of frequent heavy meals, crackling sounds in the head, changing skin complexion, an odour like that of blood in his nostrils, and a peculiar taste in the mounth. He also thought most people looked rather different and that the grass lawn had a strange bloody hue. He worried that his breathing would stop, and he was very 'clinging'.

Mr A was admitted for two weeks, when he was treated with imipramine on the assumption that he had panic reactions. He completed his undergraduate courses in the following two years without any further symptoms.

Case 2

Mr B, a 34-year-old non-commissioned military officer. He had received an injection of PP three years before presentation, to which he reacted with the intense sensation that his head had "shattered to a thousand pieces". He also described literally "seeing stars" and having a feeling of having died. He recovered in a few minutes. The doctor, mistakenly suspecting an unusual type of allergic response, warned him not to take PP again. Mr B was surprised because he was not normally sensitive to PP. Thereafter, he had two episodes of spontaneous, brief loss of touch with the environment which he described as "collapse". He reacted to any drug that produced the familiar penicillin taste (such as ampicillin) in the same way. Also any drug that altered his perception of the environment such as amitriptyline, chloroquine with promethazine, bromazepam and orphenadrine were all said to produce collapse.

Mr B was brought to our unit following a spontaneous 'collapse'. Between such episodes patient complained of crawling sensations all over the body, fatigue, giddiness, and weightlessness. He had a large family of six children from two marriages. He spoke about career frustrations and felt insufficiently catered for by military medical establishments.

Mr B was clinically depressed on presentation and responded to imipramine and psychotherapeutic support. He fully recovered after three years.

Case 3

Mr C, a 35-year-old commissioned officer, was married with two children. Eighteen months before, Mr C had received a 'usual' dose of PP on demand, as he had done many other times. This time, however, he lost consciousness before the injection was completed. He had a feeling of having died. He recalled hearing bells ringing (death knells), being on the floor when he regained consciousness shortly after, and pleading with the frightened nurse not to allow him to die. On a suspicion of malaria he was given chloroquine with promethazine. Again, he had a reaction which he thought similar but much milder.

He complained of an ever-present fear of imminent doom, death or collapse. He said he frequently checked on his body parts to ensure that they were still there. He recalled an occasion in which he dashed out of his bedroom one morning to ask his baffled wife "Where is my head? I can't find my head". On more than one occasion he had started shouting in his office, calling for help because of premonition of imminent unconsciousness He recalled blacking out several times, once while he was driving. He had since stopped driving and refrained from leaving home without a chaperon. Other complaints included headaches, dizzy spells, sleep disturbances, and suicidal fantasies. He felt better at noon and on bright days but worse in the mornings and in dull weather.

Since no prescription had given Mr C expected relief, and especially since the doctors denied any connection between his distress and PP, he increasingly turned to traditional, spiritual and 'alternative' medical sources, which was most disorganising for Mr C.

During admission, he often tried to tell between shades of morning weather to pin-point which morning most resembled the one on which he received the fateful injection. He talked about constantly playing back the sequence of events of the reaction experience "like a video".

He was admitted for a total of four weeks. Tests, including EEG, were normal. The major treatment modality was psychotherapy (individual and marital). Low doses of imipramine seemed to control his somatic distress. Compensation concerns such as being taken to the UK or USA for 'proper' medical treatment also emerged.

Case 4

Mr D is a 35-year-old senior non-commisioned officer (NCO) in the police force. Twelve months before presentation he had received two injections of PP from an unqualified medical practitioner who was also his half-brother, with whom he had a difficult relationship. After the second injection Mr D felt his head had caught fire and his body felt "showers of hot electricity". He raced around uncontrollably for one or two minutes. He felt he had a near-death experience.

Mr D had complaints of waist pain and erectile impotence for 15 years, for which no treatment, orthodox, religious or traditional, seemed to help. He also claimed that after his reaction to PP, his impotence worsened, with new symptoms including pains and crawling sensations in the head and body, sensations of his head swelling, sleeplessness, and weight loss. The reaction also marked the onset of dreams of being attacked by a "mad dog". These dreams recurred frequently and he claimed worsening of symptoms following every such dream.

Mr D was from a polygamous family. There was an unusually competitive relationship between him and his older half-brother (who had given the injection). During admission, it emerged that Mr D had a deep-rooted fear that his half brother wanted to kill him, not only to take sole possession of their joint investments but also to 'inherit' his wife, who had a lucrative retail trade.

As Mr D was clinically depressed, he was admitted for three months. His request for electroconvulsive therapy (ECT) was refused. There were compensation features manifested as an insistence on staying in hospital (which was at the expense of his employers) until he was 'completely' cured. He benefited immensely from amitriptyline and individual psychotherapy.

Case 5

Mr E, a 25-year-old clerical officer, was referred from his company clinic with notes reporting: "very hot sensations in his vertex on and off for 4 months following an allergic reaction to seclopen in our staff clinic. . . . Bouts of hot sensations in the head are associated with lack of concentration, easy fatiguability and crawling sensations in the forehead. . . . He has had courses of Largactil, phenobarbitone, Valium at one time or the other with no improvement."

The patient's recollections of the reaction to PP, given for a boil in the groin, were as follows: a strong smell of penicillin in the nostrils, ringing in the ear like a church bell, a feeling of his head being shattered, and shouting for help.

His family history was negative, although Mr E had been sickly in childhood. He improved and stopped attending the clinic after one year of treatment, which consisted of antidepressants and psychotherapy with focus on stress management.

Case 6

Mr F, a 32-year-old accounts clerk, was referred by a general practitioner who reported aggressive behaviour and incessant talk following a dose of PP and streptomycin two months previously. Grandiosity was quite marked.

Mr F was not seen by this author and his recollection of the reaction was not documented. The referring physician noted that patient was treated with parenteral chlorpromazine and intravenous diazepam, and maintained on oral chlorpromazine. The patient improved within 24 hours, wondering why he should react to PP and streptomycin, which he had taken so many times before. Five weeks later the experience recurred apparently spontaneously, with the patient singing aloud to himself claiming to have seen a great light. During psychiatric evaluation he interpreted this experience, in terms of his Pentecostal background, as a divine vision.

There was a chronic psychiatric illness in a paternal aunt. His only complaint on presentation three weeks after this episode was difficulty in concentration. He defaulted after the second visit as he was concerned about a possible psychiatric stigma.

Case 7

Mr G, 43 years old, works in a transport business, and is a retired policeman. His physician stated that he was sensitive to PP, chloroquine and lately Fansidar (a fixed drug combination of sulfadoxine and pyrimethamine).

Mr G dated his illness to 1974, 14 years earlier, when he received a one-week course of PP for testicular pain. He reacted to the last dose with tinnitus and fainting even before the injection was finished. He recalled being vaguely aware of his surroundings but totally unable to help himself. He recovered fully within 30 minutes. Subsequently, he avoided injections of all types for fear of a reaction. One year after this incident, he was persuaded to take chloroquine injection by another doctor. He soon developed tremors, palpitations, breathlessness, excessive sweating, and fainting. He remained unwell for three months, and travelled to the UK to seek further consultation with regard to possible drug reactions. One doctor, who did not believe the allergic basis of the reaction to chloroquine, treated him for malaria with the drug, keeping him in hospital briefly; there was no reaction.

In 1988, Mr G consulted a doctor for urethritis. He warned the doctor about his 'allergies'. He was given unspecified injections which he now believed to be PP because within 15 minutes he had had a severe reaction. Since then, he has had unprovoked fainting attacks. A blood pressure of 220/120 mmHg was said to have been recorded following one such attack, but this settled with bed rest. He was thereafter successfully treated with a three-week course of imipramine, until he suffered another reaction in response to the fixed drug combination.

Other complaints included decreased libido and easy fatiguability. The patient alleged being unfairly treated by the police (his previous employers), and business associates, to whom he felt much anger, as well as to his doctors.

Discussion

Only in cases 6 and 7 were the patients explicitly referred by their physicians for symptoms recognised as related to PP reactions. The patients' recall of the reaction experience can thus be distinguished from the presenting complaints. A general profile emerges as follows.

Reaction experience

(a) Immediate, intense and unexpected psychological shock which the patients associated with dying, having died or disintegration of the self.

(b) Complete or partial recurrence of the experience in circumstances containing elements of the original experience or its antecedents, e.g.:
  (i) any injection at all (cases 1–4)
  (ii) medications with any suggestive central or autonomic nervous system effects or side-effects (cases 1–4)
  (iii) weather conditions resembling those prevailing before the experience (cases 1 and 4) or suggestive of part of the reaction experience (case 2)
  (iv) unidentified environmental cues that may give the appearance of spontaneous recurrences.

(c) Symptoms diagnostically recognisable as delirium (organic mental disorder) immediately following the injection, with marked depersonalisation (expanding internally) as well as auditory hallucinations (death knells), visual hallucinations (lightning or dazzle); intense sensations of heat (case 3) and shattering sensations in the head (in three cases) are also recorded.

(d) One reaction and recurrence of typical manic features (case 6) were rather atypical. In view of family history of mental illness, precipitation of a functional illness should be suspected.

Psychiatric presentation

Apart from recurrences of the original experience, the following features were presented frequently.

(a) Symptoms of dizziness, lassitude, difficulty in concentration, insomnia, crawling sensations and heat in the head and body, depersonalisation symptoms, apprehension, fainting attacks, and agoraphobic phenomena.

(b) Preoccupation with the reaction (replaying the reaction "like a video", overinterpreting every experience to fit in with the reaction)

(c) Dreams symbolising perceived violence of original reaction (mad dog, wars); these dreams were interpreted to suit whatever explanatory theory the individual wanted to convey to the listener.

(d) Cultural elaboration of the entire experience.

(e) Better prognosis with early diagnosis and treatment.

(f) Marginal pre-morbid psychological adjustment for all seven patients as shown by the following features:
  - (i) marital stress
  - (ii) sexual guilt and conflict (fear of sexually transmitted disease, impotence)
  - (iii) career frustrations
  - (iv) excessive religiosity

It is also of interest that four out of seven of the cases came from the uniformed security professions.

The major differences between our experience and the predominant trend in the literature is that the emphasis has been on acute psychiatric symptoms, while our patients had symptoms lasting between ten days and three years. Apart from the possibility of progressing to a chronic phase, which is the main thrust of this presentation, other features detected in earlier reports but not sufficiently emphasised, included that: (a) the experience may recur within minutes (Batchelor *et al*, 1951, case 1), weeks or months (Tompsett, 1967, case 4; Menke & Pepplinkhuizen, 1974, case of the pregnant woman); (b) this type of reaction often marks the onset of other stress symptoms not previously emphasised (Mackenzie & Popkin, 1980); (c) it has been considered advisable to discontinue PP in many cases (Menke & Pepplinkhuizen, 1974) – indeed, many of the patients refused further doses (Tompsett, 1967).

Cultural elaboration and distortion

Recurrence of dreams symbolic of past trauma is widely recognised as a stress response, but there is also a cultural interpretation of dream violence as witchcraft. The recall of the dream, and any temporal relationship to the injection, may serve to focus attention on fears or deep-seated personal and family conflicts. In Nigerian popular culture injections are rated 'higher' than oral medications. Also up to 53% of prescriptions (including injections) may be made and administered by non-physicians (Mabadeje, 1974). The possibilities of non-recognition of varieties of drug reactions and delay in appropriate management are therefore further enhanced. For some patients great dissonance must have been created by failure of the modern medical assumptions to give relief, especially when the perjorative diagnostic labels of 'neurotic' and 'hysteric' were applied. Traditional and spiritual healing was sought by these patients, which led to secondary psychological difficulties.

Conclusion

Many major medical texts still omit mention of the PP reaction. Our cases have shown that chronic sequelae exist, possibly because they have not been recognised in the acute phase. The principles of treatment remain the same as for all traumatic neuroses, namely, recognition of the individual's distress, and its prompt relief by appropriate medications, usually anxiolytics. This must be done as close as possible to the time and place of the traumatic experience. In an iatrogenic trauma of this nature, recognition and explanation of the reaction to the patient in scientific terms will also help remission. It is often said that traumatic neuroses are psychodynamically devoid of meaning (Rycroft, 1968), but this does not imply that meaning may not be secondarily acquired, which appears to be the case for all our patients.

As for the issue of continuing PP after this type of reaction, Menke & Pepplinkhuizen (1974) advise avoiding PP.

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Case Conference

Resistant Bipolar Disorder

I. G. THOMAS, A. G. OSWALD and J. M. EAGLES

A 66-year-old farmer's wife had been admitted 31 times over eight years, suffering from manic depression. A variety of treatments for depressive episodes led to hypomania, and a case conference was held to discuss treatment.

Affective disorder is generally considered to have a reasonably good immediate prognosis, with episodes of illness and various prophylactic regimes maintaining euthymia. It can prove frustrating for both patient and physician when euthymia proves impossible to sustain. We present a record of a case conference held on 4 May 1988 in Royal Cornhill Hospital, Aberdeen, to discuss such a case.

As part of the Grampian Rotational Training Scheme in Psychiatry a case conference is held each week. The aim is to discuss cases of educational and clinical interest, and the conferences are attended by members of the university department, consultants, junior staff, psychologists, students, and other professionals connected with the case. The chair rotates through various consultants. The format usually consists of a case presentation, an interview with the patient (either live or on videotape), followed by discussion.

Objectives

The principal aims of the conference in question were to consider the treatment efficacy to date, and to suggest future treatment possibilities for a lady with treatment-refractory manic–depressive illness.

Presentation of case

Psychiatric history

Mrs G is a 66-year-old married lady whose first presentation to the psychiatric services was in 1954 with a diagnosis of puerperal depression after the birth of her first child. She had incised her abdomen and perforated her small and large bowel with a pair of scissors. She responded well to electronculsive therapy (ECT). She was again admitted and responded to ECT following the birth of her second child in 1955. The birth of her third child did not precipitate a recurrence, and there was no further contact with psychiatric services until 1973, when she was admitted in a hypomanic state. She had three admissions in 1973 and was commenced on lithium. She had a brief admission in 1978, a two-month admission in 1979, then 31 admissions over the next eight years, usually lasting one or two months.

In 1980 she was commenced on flupenthixol (1 mg b.d.) for 18 months. In 1983 she was commenced on carbamazepine for one year; this was discontinued after a number of falls and a Colles' fracture. Lithium was restarted in 1984, and in 1985 carbamazepine was reintroduced in combination, but discontinued after more falls. Carbamazepine blood levels were 7–10 mg/l and lithium 0.2–0.9 mmol/l. She has been on thyroid replacement therapy since 1981. Episodes of depression have been treated with a variety of antidepressants: amitriptyline, nortriptyline, dothiepin, clomipramine/L-tryptophan/lithium combination, and ECT. These were usually discontinued shortly after improvement, owing to the onset of hypomania.

Admissions were usually preceded by a period when the patient would remain in bed, sometimes for eight weeks, and occasionally they would be preceded by overactivity and irritability. Between admissions her mood is not stable; most of the time she is reported to be depressed, doing very little for herself, then there are sudden episodes when she will arrange everything, talk to everybody, and drive around the countryside (occasionally deliberately on the wrong side of the road). When hypomanic she often makes extravagant suicide threats, although there has been only one recent parasuicide, by overdose, in 1982. There is no suggestion of abuse of alcohol or any other drug.

Personal history

Mrs G was the second of five siblings. Her father was a farmer and she has lived all her life in the farming community of Aberdeenshire.

She had an unremarkable birth, infancy, and childhood. She reports that she was a happy child and an average scholar, leaving school at the age of 16 to work on the family farm. She was engaged briefly at the age of 20, but remained with her parents until marrying her present husband (also a farmer) at the age of 30. It appears that her marriage was at first happy and stable, but naturally it has been considerably disrupted by her many years of mental ill health.

During periods of well-being, she has worked as a 'farmer's wife', coping well. She has one son, who lives at home and will take over the farm when Mrs G's husband retires, and two daughters, one of whom lives on a nearby farm and one who is a nurse.

Family history

Her father committed suicide by hanging at the age of 53, when Mrs G was 16. He had not had contact with psychiatric services. Her mother died aged 57 from heart disease. Her elder brother died in 1985 in a long-stay psychiatric ward. He was diagnosed as schizophrenic.

Previous medical history

The only medical history of note is hypothyroidism, treated with thyroxine since 1982. Other blood parameters are within normal limits, and there is no evidence of neurological abnormality on physical examination.

Pre-morbid personality

Mrs G was described as friendly, capable, and intelligent, an 'ordinary housewife' who managed the farm finances competently. Now her husband reports 'endless swings', with only two months of near normality in the past two years.

Mental state examination

Mrs G's mental state obviously fluctuates greatly. She looks rather older than her years, but has no abnormal movements or mannerisms. On the day of the conference she had just returned from a visit home and was mute but fully conscious; she was eating nothing, drinking only with persuasion, and refusing medication. At times she becomes preoccupied with her financial situation (which is healthy), but she has not suffered from frank depressive delusions. Hallucinations have not been documented. Recent testing has not uncovered any deficits in her cognitive functioning.

Interview

The interview was recorded on videotape two weeks before the conference. At that time Mrs G had been in hospital for 15 weeks and had been treated with lithium and thyroid replacement. She had stopped her lithium before admission. During her admission she had slowly improved. The following comprises excerpts shown to the case conference.

DR THOMAS (*registrar in psychiatry presenting case*): How old were you when you first had difficulties?

MRS G: The first time that I can mind was when my son was born, antenatal depression or something, but I dinnae mind a'thing about it.

DR THOMAS How old would you have been?

MRS G. Just over 30; 32 or so.

DR THOMAS· Had you had any problems with taking to your bed before then?

MRS G· No, this taking to my bed wasn't to start with, it was after a while.

DR THOMAS So after your son was born you went into hospital for a bit but you can't remember a lot about that.

MRS G No, I cannae mind. I was in the infirmary at first, then I was in Kingseat, but I cannae mind a lot about it; there's a lot of years I cannae really remember a lot about. The girls going to school – it must have been when the girls were old enough to start getting the meals that I started going to my bed.

DR THOMAS. Can you remember what you were like at the time you came into hospital this time?

MRS G Dr McCance [the patient's consultant] she said, "You're on the go again".

DR THOMAS. I know that in the past you've had times when you've been on the go too much, and other times when it's been the opposite problem. Do you notice the difference between the two?

MRS G I ken myself that I go like that, but I don't think I realise that I'm going too much, as I say when I'm doing too much, I've nae feelings for anybody. My husband's inclined to tell me that I can tell a'body what to do, and I suppose that's right enough because he doesn't say things that aren't true.

DR THOMAS When you're feeling down, do you tell people what to do then?

MRS G No, no, I'm not telling then, I maybe think, just lying there. If you're just lying there for weeks, your thoughts are nae right thoughts, something comes up, and it's all out of proportion, and I suppose sometimes I dinnae drink enough or eat enough, and I come in here not feeling well because I've not been eating or drinking and that's why I say it's maybe up to myself to do more. Mercy, Dr McCance has done a'thing she knows.

DR THOMAS What's the longest length of time you've been in hospital?

MRS G This time. This is about three months – I don't think I've been in that long.

DR THOMAS. And over the past two to three years, what's the longest length of time you've been at home?

MRS G: Let's see, maybe a month, something like that; a month functioning properly, and then weeks in my bed. I think if I remember right Kathy Urquhart [the community psychiatric nurse] said I'd been eight weeks in bed. My husband just left me there. Well I dinnae ken. I suppose I was ill.

DR THOMAS Say five or six years ago, were you keeping better then?

MRS G: Aye, aye.

DR THOMAS As you know, one of the things we're having to think about is how we can keep you well for longer than a month, and, as you say, the time you're keeping well for seems to be getting shorter. Have you any ideas yourself?

MRS G: No. I think it needs to come from myself, kind of control myself better, and if I see something that bothers me, dinnae let it bother me, leave the rest of them alone. You ken what I mean? They've their own worries and let them alone. Just try and keep myself on an even keel.

DR THOMAS: I know Dr McCance has mentioned the idea of an operation, a brain operation. Have you had any thoughts on that?

MRS G· It's an awfy thought. Ach, I'm 66, and no, I dinnae think so. I'd like to do it more myself.

Conference discussion

DR OSWALD (*chairman, consultant psychiatrist*): Are there any comments on the video? There was one thing that struck me – when she was well she had no insight that there was an endogenous component to her illness, that she seemed to think that if only she could cope better with her environment then she would be better.

DR TROTTER (*consultant psychiatrist*): I wonder if that's the case. I don't think that she was euthymic – I thought that she was still mildly depressed, and that she was being self-deprecatory.

DR McCANCE (*Mrs G's consultant*): I think you're right, but her condition was relatively good.

DR TROTTER: Has her medication been changed in the last three weeks?

DR McCANCE: She's been out on pass and stopped taking her medication and been in her bed. She remains well now for a matter of days before she's depressed.

DR THOMAS: In a part of the video that I did not show she said quite specifically that she stops taking her medication when she starts getting depressed, not the other way round.

DR EAGLES (*consultant, psychiatric tutor*): Can we assume that her compliance with medication while she's in hospital is what we think it is?

DR THOMAS: Her lithium levels while in hospital are reasonable, although as an out-patient they vary widely, as do the thyroid tests.

DR OSWALD: I wonder if it might be worth thinking more about that, since the only study that has suggested that thyroxine is effective in the prophylaxis of manic-depressive illness (Stancer & Persad, 1982) also suggested that it tends to be particularly effective in post-partum illnesses, so there may be a link there. That was a small study; they looked at ten patients refractory to lithium prophylaxis and found that by adding thyroxine, five out of ten were reasonably well maintained.

DR EAGLES: I think the important thing about their study was that thyroxine was not just returning patients to euthyroid levels – you had to give them three or four times the normal replacement dosage, 0.3 or 0.5 mg per day. There is also evidence that rapid cyclers are more prone to become thyroid deficient on lithium than are non-rapid cyclers (Cho *et al*, 1979; Cowdry *et al*, 1983) and that's odd. There must be some link between thyroid and rapid cyclers.

DR OSWALD: Probably everyone accepts that lithium has very little effect in up to a quarter of bipolar patients (Dunner *et al*, 1976) and particularly rapid cyclers tend to respond poorly (Roy-Byrne *et al*, 1984). That certainly seems to be the case with this lady if we define rapid cyclers as people with three or more affective illnesses in one year. Even when lithium does not prevent affective illness, it can often reduce the severity and/or frequency of the illness, and although that does not seem to be the case recently, there is a suggestion that it did in the early stages.

I wonder whether we have given up on carbamazepine too quickly, and whether we have been trying to get the carbamazepine levels too high. Most reports have assumed that we ought to reach the sort of levels we need in epilepsy (Okuma *et al*, 1981) but it may be that much lower blood levels have a prophylactic action.

PROFESSOR ASHCROFT (*professor of mental health, Aberdeen University*): Whatever drug you use there is going to have to be some attempt to tackle the compliance problem.

DR CALDER (*senior registrar in psychiatry*): Would there be grounds for compulsorily admitting this lady for say six months, to see the pattern?

DR TROTTER: This lady's illness seems to fall into four decades. In the 1950s she had two serious episodes of depression with biological precipitants in the form of childbirth, then she disappeared in the '60s, but came back in the '70s although by and large maintained on lithium, and then things began to worsen in the 1980s, and I wonder if we can see what has happened to her in the past eight years, not as a series of depressive relapses, but as one continuing depressive episode that we need to tackle properly, even if it means detaining her.

DR McCANCE: The difficulty with having her in hospital is that her husband is unwilling to agree because she will hold it against him.

DR MATTHEWS (*registrar in psychiatry*): Looking at it from the four separate decades, has there been some significant change in her home life or social circumstances that is continuing to precipitate episodes of depression?

DR McCANCE: She moved out of the main farmhouse, and the son moved in, but I do not think that caused any great trauma. She inherited quite a lot of money, and that caused some problems. Her children are doing very well.

PROFESSOR ASHCROFT: Could anyone make a case for stopping all drugs and seeing what happens? At least then you start off on a baseline and you can take it from there.

DR EAGLES: I would support that because I don't think over recent years there is any evidence that lithium has helped her and certainly Wehr *et al* (1988) suggest that lithium can occasionally make rapid cyclers worse, and there are many reports in the literature of tricyclic antidepressants also making them worse (Wehr & Goodwin, 1987).

DR CALLENDER (*consultant psychiatrist*): There are reports of relapse being precipitated by lithium withdrawal (Mander & Loudon, 1988). This lady has repeatedly been in episodes of relative or absolute lithium withdrawal and although in retrospect it is difficult to say whether this caused relapse or vice versa, perhaps it would be better to withdraw it.

PROFESSOR ASHCROFT: There are drug therapies that she has not had. Monoamine oxidase inhibitors (MAOIs) are worth considering. The other thing which she's not had which other people would try in rapid cycling patients is valproate.

DR McCANCE: The other thought I had is to use intramuscular flupenthixol, to overcome possible noncompliance.

PROFESSOR ASHCROFT: The other thing about rapid cyclers is that sometimes they benefit from a change of environment, or even a change of ward. It is nothing whatever to do with the standard of care on a ward, but a switch of people looking after them seems to sometimes produce a change. Also there are one or two units that specialise in the treatment of resistant depression, for example Professor Eccleston's in Newcastle, and if someone has as bad a history as this, perhaps we should be thinking of asking them if they would take her. They are used to treating this sort of illness; they would run through the different therapies, and would not take her for just a short time. They do claim a high success rate, but it is very difficult to run controlled trials on this sort of patient.

DR OSWALD: I think they would be faced with exactly the same problem. Could we ask Stephen Bell here whether there is anything the psychologists might offer to improve a patient's compliance.

MR BELL (*top-grade clinical psychologist*): In such patients, I don't think so.

DR MATTHEWS: Does it not strike anyone as a little odd that after having recurrent illnesses, on the evidence of the video, she has no insight whatsoever that she can be helped in any way by physical treatment.

PROFESSOR ASHCROFT: To a certain extent, she is right. I think that starting afresh may be our only hope of changing attitudes. I would push for that and I would not feel a failure in doing so.

DR OSWALD: I wonder if there is nothing more we could do locally to improve her compliance. At our day hospital we have a group we refer to as the 'Health Talks Group', and one of its main functions is talking about taking your pills.

DR McCANCE: She lives a long way from the hospital.

DR CALLENDER: It's important to remember simple things like one-day dosing where possible, avoidance of multiple medication, and so on.

DR CALDER: She will not be more compliant unless we increase her insight. Surely we have to take control of this lady in a formal way, with a second opinion, so we can monitor whether she takes her tablets, and if she does not and she becomes ill, then we can say it is through non-compliance. At the moment it is only assumption.

DR McCANCE: Not entirely – she has relapsed in hospital on tablets. She does take them when she is in, but whether they are the right tablets is of course questionable.

DR OSWALD: I was hoping to discuss whether psychosurgery might be an appropriate intervention.

DR TROTTER: I do not think that you have been able to prove that she does not respond to a variety of treatments. I think she would be sent back to you fairly quickly by any psychosurgeon saying try A, B, C, D, and E first.

DR THOMAS: There is the report by Poynton *et al* (1988) suggesting that psychosurgery can be used to ameliorate manic–depressive illness.

DR McCANCE: There have been two reports of nine cases (Lovett & Shaw, 1987; Poynton *et al*, 1988), not generally startlingly good, but occasional ones *have* been startlingly good: 22% very good results in bipolars, 67–70% in unipolars, over a four-year follow-up in the bipolars. I did send a bipolar lady for stereotactic subcaudate tractotomy and she has done remarkably well, so that obviously has an effect on me. In the bipolar ones it seems to control manic spells better than depressive episodes, which was contrary to what had been thought by Pippard (1955).

DR OSWALD: Certainly we could not proceed without her full approval, and she seemed very reluctant. MAOIs are an interesting suggestion – people have tended to use clorgyline rather than phenelzine, a type A MAOI. It has fewer side-effects but it too has been reported to induce mania in a substantial minority of patients. Maybe we can turn to Dr McCance and ask whether she has gained anything from this discussion.

DR McCANCE: I think it is extremely helpful to hear other people's ideas. I think the thyroxine is well worth trying. I am a little doubtful about the MAOIs, having seen her high so often. Valproate seems worth a trial and depixol by intramuscular injection is another that I would consider. I would also consider her going to somebody else's ward, but have known her for a very long time, so I have a certain affection for her, and I think she has for me. Finally, I think I probably should admit her compulsorily. I want to be able to keep her in hospital, although for somebody who has been ill so much I feel if she has a few days when she feels well it seems a shame to keep her in hospital.

Addendum

After the case conference Mrs G was detained under Section 18 of the Mental Health Act (Scotland) 1984 and has been in hospital for one year. Her lithium was discontinued and she had been tried without medication other than thyroid replacement and on intramuscular flupenthixol. Her mood remains predominantly depressed with short episodes of near euthymia. Her willingness to comply with prophylactic medication remains unchanged. Her mood has been such that it is not felt that she could have been maintained out of hospital.

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Anorexia Nervosa in Adolescents of Asian Extraction

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Case reports of anorexia nervosa in subjects of Asian (Indian subcontinental) extraction have been rare. Three Asian adolescents with anorexia nervosa are described, one of whom is male. The impact of the Muslim festival of Ramadan is described in the two Muslim cases. There is a need for better understanding of attitudes towards body shape and food within the subculture.

Recent publications not only point to an increase in the number of cases of anorexia nervosa over time (Szmukler *et al*, 1986) but also to the occurrence of the condition in non-white populations in the UK (Buchan & Gregory, 1984; Thomas & Szmukler, 1985; Holden & Robinson, 1988). The majority of case reports of non-white anorectics and bulimics have been of subjects of Afro-Caribbean extraction.

Kendell *et al* (1973), surveying the case registers in London, north-east Scotland, and Munroe County, New York State, noted one case of a student nurse from Guyana of Asiatic Indian stock. More recently Lacey & Dolan (1988) described an 18-year-old woman of Pakistani extraction and a 26-year-old woman of Indian African parentage, both of whom had bulimia.

Reports of anorexia nervosa in adolescents of Asian (Indian subcontinental) extraction are rare, especially in males. The three cases described met the DSM–III–R diagnostic criteria for anorexia nervosa (American Psychiatric Association, 1987).

Case reports

Case 1

Miss J, a 15-year-old pre-pubertal Muslim girl, was referred to the Regional Adolescent Unit in Birmingham from a local consultant paediatrician. The patient had been diagnosed and treated as an in-patient in a paediatric ward but had relapsed on discharge.

The eldest of four children and an only girl from a working class, nuclear, Muslim family of Pakistani origin, the patient was born in Pakistan and came to the UK when five years old. She had always been a quiet, shy, and conforming girl, and a hard worker at school.

At presentation her height was 148 cm and weight 29 kg (24% below ideal on height and weight standard chart). She was a small, emaciated girl who had lanugo hair and peripheral cyanosis.

Although a vegetarian since eight years of age (since she felt that eating meat was cruel), her dieting to lose weight started when 12½ years of age. The latter was temporally related to a serious rift between her father and her maternal grandparents. Both the patient and her mother were particularly close to the maternal grandparents. The rift was caused by the mother's brother divorcing his wife, who was the father's niece. Father then felt obliged to shun contact with the mother's parents. It was interesting that the maternal grandparents initiated the first family practitioner consultation for the patient's loss of weight. After the onset of the illness the maternal grandparents' contact with the family increased.

At onset the patient felt she was too fat and started dieting, and she gradually lost weight. She became overactive and showed increased interest in cooking. The patient was 14 when admitted to a general paediatric unit. During three months she gained weight from her admission weight of 26.5 kg, to reach 32.6 kg at discharge and continued to attend the hospital school; she ate well at school but not at home. Her weight continued to increase, to peak at 35 kg. She lost weight on return to her own school, and this accelerated over the Ramadan fast. She also began vomiting after the big meals during Ramadan.

The patient was referred and admitted to the adolescent unit as an in-patient. Her admission weight was 29 kg. Although she had considerable fears of getting fat and body-image disturbances, she complied with the ward anorexia regime with little protest. As she gained weight and underwent individual psychotherapy, her psychopathology improved. Attempts at family therapy were abandoned because of parental reluctance. It had proved possible to negotiate exemption from fasting for the patient during the next Ramadan. However, the patient felt it necessary to eat in secret. Her weight on discharge from the adolescent unit was 38.05 kg, which was her ideal weight based on Tanner–Whitehouse tables.

At six-month follow-up, the patient's weight was within 15% of her ideal weight and she had attained her menarche. At five months post-discharge the patient was admitted to a general hospital following urinary retention. Haematocolpos due to an imperforate hymen was diagnosed – incision and evacuation was followed by a normal period. Over the following seven months, the patient had irregular and scant menstruation, the last menstrual period being eight weeks before last follow-up. Her weight had fallen very gradually over the latter period to fluctuate between 33.85 and 34.20 kg. There was no history of bulimia.

Case 2

Mr S, a 16-year-old post-pubertal Sikh boy, was referred to the Regional Adolescent Unit from a child guidance centre.

The second of four boys from a middle-class, nuclear, Sikh, Kenyan Asian family, the patient was a friendly and conscientious boy who had always been neat and tidy. He was born in Kenya and came to the UK as a baby. At school he was considered to be of average ability, while his three brothers were bright. He was a very hard worker at school and was in the top group in all subjects. There was pre-morbid history of obesity (26% above ideal weight). At presentation his height was 171 cm, and weight 44 kg, (24% below ideal and 40% below best weight). He had lanugo hair, bradycardia, peripheral cyanosis, and his oral temperature was 36.3°C. He was described as withdrawn and morose on admission.

Within the family the patient and his dominant father were in conflict over the patient's wish to get his hair cut (Sikh's are expected not to do so). There was a suggestion that the father was somewhat distant until the eating problem started. The patient was, however, generally able to get his own way with his mother.

The onset was eight months before presentation and immediately following two events: a teacher remarked that the patient was fat; and the patient wanted an expensive leather jacket which the father said he could not afford to buy. The patient started dieting to lose weight and to save money for his leather jacket. He bought and cooked his own food, ate on his own, was overactive, and did extra work around the house for extra pocket-money. He lost 23 kg in six months. In the few weeks before admission he had become withdrawn, irritable, and rarely laughed. He was caught shop-lifting one month before admission.

At admission he was adamant that he was too fat and was preoccupied with his body shape and food. He denied any problems and was very reluctant to participate in the anorexia regime and was discharged when he absconded home (weight 47 kg). Readmission under the Mental Health Act was not pursued for the following reasons: the parents were definite that they wanted to carry on the ward programmes at home; the patient's nutritional status had improved and he was not dehydrated; the depressive features noted in the history were felt to be secondary to conflict with parents; and the patient's family practitioner was happy to carry out close monitoring of the patient's nutritional status. He was lost to follow-up.

Case 3

Miss A, a 15-year-old post-pubertal Muslim girl, was referred to the Child Psychiatric In-Patient Unit, at the Children's Hospital, Birmingham. The patient had previously been treated as an in-patient in a paediatric ward in a general hospital, where she lost weight – interestingly this coincided mainly with Ramadan.

The younger of two children and the only girl from a nuclear, working-class, Pakistani Muslim family, the patient was born in Pakistan but has lived in the UK since eight years of age. There was history of pre-morbid obesity in the patient (best weight 30% above ideal).

It was felt that the patient was rather spoilt and able to manipulate her parents readily. Her only sibling, an older brother, lived away from home and had only irregular contact with the family. The mother had no relatives in the UK and was socially somewhat isolated. The patient was close to her parents and had, in fact, said that she would stay single so that she could stay with her parents.

At school, she was considered to be of average ability and 'normal' for her age and culture.

At presentation, her height was 156 cm and weight 34.5 kg (30% below ideal). She had amenorrhoea having attained menarche when 12 years old. She had lanugo hair and bradycardia.

The onset was eight months before admission and was immediately preceded by being ridiculed for being overweight. The patient started dieting and exercising with increasing severity and over about three months had lost 13 kg. She lied about food and eating, was overactive, increasingly cooked for others but mostly ate on her own. Her problems worsened over Ramadan and she began vomiting. She was admitted to a general paediatric ward for three weeks when she was suspected of vomiting, bingeing, waterloading, and exercising. Her weight fell from 34.5 kg to 33 kg, and she was referred for admission to the Child Psychiatric Unit. She was convinced that she was still fat and was very unhappy at the ward regime – she continuously pestered her parents for a discharge. The patient was discharged against medical advice (five weeks after admission), ostensibly to go to Pakistan since her paternal grandmother had died. Her discharge weight was 41.75 kg.

She was seen two months later as an emergency, after repeated suicidal attempts, dieting, bingeing, vomiting, and tearfulness. She had, in fact, not left the country, the account of the grandmother dying being a ruse. She was referred to the adult psychiatric services because of a lack of beds.

Admission to an adult ward was advised but she refused to stay in a 'mental home'. Detention under the Mental Health Act was not considered appropriate for the following reasons: her weight was 44.35 kg; it was considered that she was not significantly clinically depressed and that her attempted suicides were manipulative rather than serious attempts to kill herself. The patient and her mother returned to the adult unit within two weeks with similar complaints but again declined admission.

The patient is being followed up as an out-patient by the child psychiatric team. A trial of antidepressants was commenced which the patient stopped after two months, when she failed to keep several appointments. When she attended for review after another two months (nine months after admission to the Child Psychiatric Unit), her mood had improved, and she was no longer bingeing or vomiting. At follow-up ten months after admission her weight was 48.2 kg (1 kg below ideal).

Discussion

It has been postulated that where anorexia nervosa occurs in low-risk groups, there might be evidence of greater loading of factors thought to cause or facilitate the condition (Thomas & Szmukler, 1985). Pre-morbid obesity might be such a factor (Holden & Robinson, 1988).

Of the several factors noted by Robinson & Anderson (1985) in black subjects (e.g. male sex, serious disturbance before onset, family history of physical conditions including obesity), Holden & Robinson (1988) found support for parental separation in their series, although the majority of the black subjects in the latter series were bulimics. Since the above findings were from a series of subjects of Afro-Caribbean extraction and the present series of Asian patients is small, one needs to interpret the comparisons with caution. Nevertheless, it is interesting that one of the three Asian patients was a male and two of the three had history of obesity. There was no history of serious psychological disturbance or of parental death or separation. The clinical features themselves do not appear to be particularly atypical.

It is heartening to note that in spite of the illness occurring in a low-risk group, two of the three adolescents presented to a specialist unit within a few months of onset.

Of particular interest are the two Muslim girls whose condition worsened over the Muslim fast of Ramadam. During this lunar month, which tends to fall about ten days earlier each year, all Muslims are required to fast from dawn to dusk (as determined by the local priests) but may eat during the hours of darkness. Traditionally, the end and the beginning of the fast are marked with a rather rich and sumptuous meal. In the UK, when Ramadan occurs during the summer months, the fasting period is long and there is often one single large meal during the hours of darkness instead of the usual two or more. The breaking of the fast begins with prayer and the meal itself is a ritualised family occasion. Thus, this period presents Muslims with a socially accepted and even expected period of fasting followed by pressures to consume rather large quantities of food. A worsening of the condition in terms of weight loss was reported in both the Muslim patients and they both vomited after the meal over Ramadan. Support for the interaction between this cultural/religious ritual relating to food and eating disorders comes from the report of an 18-year-old Pakistani Muslim bulimic whose condition started over Ramadan (Lacey & Dolan, 1988). With one of the patients reported here, it was possible to negotiate an exemption from fasting with her family. Although this patient managed not to fast over this period, she expressed considerable distress at having to eat in secret. Not surprisingly, the patient needed considerable support.

Of the hypotheses put forward to explain the rarity of anorexia nervosa and bulimia nervosa in non-Western cultures, the most likely one appears to be that these cultures value a slim body shape much less than Western, white cultures. Nasser (1988), reviewing the research into the relationship between these syndromes and culture, concluded that adoption of Western cultural values by other societies is associated with the emergence of similar syndromes in their cultures. Relevant to the occurrence of anorexia nervosa in Asians living in the UK is Furnham & Alibhai's (1983) study comparing the perceptions of body shape in three groups of females. These were Kenyan Asian, Kenyan Asian British (living in the UK for at least four years), and white British females. They found that the Kenyan Asian females rated larger body shapes more favourably than the other two groups. The Kenyan Asian British group's perceptions were similar to the British white females' perception of body shape. Nasser (1986) compared two matched groups of female Arab students attending London and Cairo universities and identified six cases of bulimia in the former group, lending support to the view that exposure to Western values is a strong pathogenic factor in these syndromes.

From the increasing number of reports of anorexia nervosa and bulimia in minority non-white groups within the Western culture, it would seem prudent to expect this trend to continue. Hence, there is a greater need for attempts to understand attitudes towards food and eating within these subcultures. Some of these attitudes and values are likely to be of direct relevance in the treatment of eating disorders.

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Pica in a Patient with Anorexia Nervosa

I. J. McLOUGHLIN and F. HASSANYEH

A case of an unusual pica in a patient with anorexia nervosa is described. The patient was also found to have iron-deficiency anaemia. The relationship of mineral deficiency to pica and anorexia nervosa is discussed.

Pica can be defined as the compulsive eating of unusual substances. It takes many forms and can be cultural and non-pathological; in its severest form, however, it can result in death or serious physical pathology (McLoughlin, 1988). Physicians have been aware of pica for centuries. It is probable that pica does not delineate a specific disease entity but that it is a symptom which can occur in a number of varied physical and psychological conditions (McLoughlin, 1987). Reports have described cases of pica in association with deficiencies of trace elements, particularly iron and zinc. There are, for example, many cases in the literature which suggest that the compulsive eating of ice (a specific type of pica known as pagophagia) is linked with iron-deficiency anaemia (Coltman, 1969; Moss *et al*, 1974). Some forms of pica are also associated with mineral deficiency due to the chelating action of the pica substance, this particularly occurring in earth-eating picas. Ice eating could not, however, result in mineral deficiency. Coltman (1969) investigated pagophagia in 25 women who were all subsequently found to be suffering from iron-deficiency anaemia. In all cases the pagophagia was cured within two weeks of parenteral iron injections. Reynolds (1969) studied 38 consecutive cases of patients with iron-deficiency anaemia, two-thirds of these patients admitting to ingesting 500 ml of ice or more per day. In 22 out of these 23 patients the symptoms disappeared following treatment of the iron deficiency with oral or parenteral iron.

Case report

Mrs A is a 51-year-old married housewife who lives with her husband and nine-year-old daughter. She has a nine-year history of psychiatric disorder which was precipitated by the birth of her fourth child. The child was premature and severely ill, requiring frequent admissions to hospital and blood transfusions for haemolytic disease of the newborn. Shortly after the birth, the patient's mother died and Mrs A developed symptoms of depression and anxiety. Retrospective diagnosis of the depression according to DSM–III–R criteria (American Psychiatric Association, 1987) showed this to have been a dysthymic disorder (depressive neurosis). In view of associated symptoms of anxiety and difficulty sleeping she was treated with amitriptyline. In addition, she also received regular counselling and supportive psychotherapy. She was discharged much improved after two years of treatment.

Three years ago Mrs A's appetite gradually decreased, and her weight decreased to 39 kg (more than 20% below average weight for a woman of her age, and for her height of 1.675 m). She had distorted body image and an intense fear of becoming fat. She felt a need to purge herself of food and resorted to laxative abuse, taking more than

50 Senokot (senna) tablets a day. She did not however induce vomiting. After losing weight the patient also developed amenorrhoea. These features conform to a DSM-III-R diagnosis of anorexia. On the Eating Attitudes Test (Garner & Garfinkel, 1979) she had an abnormally high score of 94. Mrs A informed us that her preoccupation with weight and shape may have been precipitated by being told that she could put on weight after discontinuing a diuretic which had been prescribed by her family doctor for 'puffy ankles'. The cause of her 'puffy ankles' is uncertain, but a review of her medication does not reveal that she was receiving treatment (such as a monoamine oxidase inhibitor) which could have caused this. Nor did she have a history of pre-menstrual tension, which is known to cause ankle oedema.

The patient is the youngest of three sisters and one brother. She describes a happy childhood and denies difficult or painful childhood experiences. She stated that she enjoyed school and had many friends. She admitted using laxatives regularly as a child for chronic constipation. Mrs A describes her marriage as being very happy. It is clear, however, that her marriage is dysfunctional and her illness appears to maintain a good deal of support from her husband. The unplanned birth of her daughter late in her marriage is probably significant. The child's illness meant that Mrs A did not gain the attention she had formerly received. She proved resistant to any psychotherapeutic approaches. There are two adult sons from the marriage and two grandchildren. The patient lives in a remote isolated rural community. She complains of feeling socially isolated but tends to avoid social contact.

Since developing anorexia Mrs A has become increasingly concerned about the food she eats and her diet has become increasingly restricted and bizarre. Initially she would avoid eating all foods other than fruits or soups. Over the last two years, however, she admits to eating up to 40 ice cubes a day; this habit continues all the year round, the patient having no idea how it started. The ice is allowed to melt in her mouth without being chewed, the patient regarding it as "a refreshing drink without much volume of liquid". It is interesting to note that the patient complains of soreness in her mouth, lips, and tongue, and that sucking ice is seen as a way of relieving this discomfort.

She has also started to freeze soft fruit, which she eats iced, deriving much pleasure from the small amounts of food. She now says that she would not eat these foods uniced, and she becomes upset when none is available. She has also over the last few years developed a craving for lettuce, raw cabbage, and watercress, which she likes because they contain few calories; her craving for lettuce is such that she becomes "fanatical" if she runs out. Mrs A denies that she had the habit of eating ice or frozen food during childhood or pregnancy or of ever having known anyone with a similar habit. It is interesting to note that soon after the patient developed this unusual habit her haemoglobin level was found to be low at 9.4 g/dl. Her folate and vitamin B12 were normal but her serum iron was low at 8 μmol/l (normal range 11–29 μmol/l) and serum transferrin was 2.96 g/l (normal range 2–4 g/l). Her serum zinc was 10.2 μmol/l (normal range 9.5–17 μmol/l).

We attempted to treat Mrs A with oral iron, but after developing nausea soon after taking it she refused further treatment. During follow-up her haemoglobin improved, currently being 12.1 g/dl; her serum iron, however, remains low at 8 μmol/l, serum transferrin begin normal at 3.85 g/l. She continues to eat ice. She also continues to show features of anorexia, even though her weight is now 47 kg. She remains preoccupied with her weight and shape and continues to abuse laxatives. Her most recent EAT score is 89 (the cut-off point for abnormal eating is 30). The most recent Beck Depression Inventory score is 14 (Beck, 1978).

Discussion

It is highly probable that Mrs A's iron-deficiency anaemia was secondary to her poor dietary intake, and it is well known that iron-deficiency anaemia can cause stomatitis. It is of interest to note that Mrs A's habit of sucking ice developed not long before her iron-deficiency anaemia was biochemically diagnosed. We feel it is probable that there is a link between them; indeed, one of the reasons which Mrs A gave for her sucking ice was to relieve the symptoms of her stomatitis.

Crosby (1976) asserts that pica occurs in more than 50% of patients with iron-deficiency anaemia, with half the patients eating ice. He believes that the discovery of pica may be the first clue to iron deficiency and can occur before the patient is severely anaemic. Deficiency of zinc may be considered for Mrs A's pagophagia, reports in the literature having linked zinc deficiency with both pica and anorexia nervosa (Arcasoy *et al*, 1978; Baken, 1979). In Mrs A's case, however, serum zinc was in the normal range, although Grant *et al* (1988) indicate that serum zinc may be a poor indicator of total body zinc.

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Left Temporal Lobe Damage in Asperger's Syndrome

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In the search for neurobiological factors in the aetiology of autism, interest has focused on the temporal lobes. We present a case of Asperger's syndrome in an otherwise healthy adult with direct evidence of left temporal lobe damage on computerised tomography.

Neurobiological factors are generally accepted as being of aetiological importance in autism (Rutter, 1974). Such factors are diverse and include biochemical (Gillberg *et al*, 1983), electrophysiological (Skoff *et al*, 1980), and structural brain abnormalities (Bauman & Kemper, 1985; Prior, 1987). There has been particular interest in the role of temporal lobe damage (Hetzler & Griffin, 1981). Magnetic resonance studies have noted fourth-ventricle enlargement but have been unable to localise this to structural pathology (Gaffney *et al*, 1987), and computerised axial tomography studies have been inconclusive (Creasey *et al*, 1986). Much of the other evidence is inferred from indirect neuropsychological studies (De Long, 1978). Both Wing (1981) and Rutter (1974) have stressed that, while there are clear differences, there is a relationship between higher-functioning autism, autistic psychopathology, and Asperger's syndrome in adults. They consider these conditions to lie on a biological continuum with infantile autism. Thus isolated areas of brain pathology in the more specific Asperger's syndrome may give clues to the localisation of pathology in the autistic syndrome as a whole. We present direct evidence of temporal lobe damage in a case of Asperger's syndrome.

Case report

A 34-year-old Caucasian man was referred for investigation of short periods of inaccessibility and episodes of disturbed behaviour. There was no family history of psychiatric disorder. His mother reported a difficult forceps delivery due to oblique lie, at full term. As a baby he disliked being picked up and was never as affectionate as his sister, born two years later. Speech remained merely a babble until age two and half years when he said his first words in the form of a sentence, "No bulb in dat light, there". Other milestones were normal. As a toddler he neglected toys but would push kitchen pans up and down the kitchen floor for long periods.

He disliked being in groups of children at primary school but was described as a good pupil and his parents regarded him as normal. His interests were astronomy, astrology, and reading the *Guinness Book of Records*. He passed the 11+ examination, attended grammar school, and was well placed in all subjects, winning a prize in Latin. He developed an interest in Esperanto and his family first had cause for concern when, age 13 years, he began to spend many hours alone trying to invent a new language. Previously placid, he began to be argumentative and to have temper tantrums. He neglected his school work but avidly read books on numerology and astronomy. He was bullied at school and became isolated and withdrawn. After two years his school performance was hopeless, his family found communication with him impossible, and he was referred to a child guidance clinic. There was no evidence of organic impairment or psychotic phenomena. Psychometry revealed highest scores on rote memory. A diagnosis of possible schizophrenia was suggested and neuroleptic medication prescribed in an effort to control the behavioural outbursts.

For the past 17 years the condition has been relatively stable. The patient lives at home with his mother, with close attention from community psychiatric services. He continued to take neuroleptics up to the time of referral. He is a dishevelled though pleasant man who spends many hours alone reading astrological texts, books on chess, and the *Guinness Book of Records*, from which he can quote many statistics. He makes virtually no eye contact and dislikes crowds intensely. He has little spontaneous speech although answers questions appropriately and can discuss astronomy and chess with enthusiasm. Often he will walk off in the middle of a conversation. There is no evidence of any mood disorder or of abnormal thought processes and he denies both delusion and hallucination. He appears to have no insight into his abnormal social interactions. Physical examination reveals a mild thoracic kyphoscoliosis and no focal neurological signs.

A computerised tomography (CT) brain scan (Fig. 1) revealed low density in the left temporal region and atrophy of the left temporal pole. Other brain areas were normal. Resting electroencephalography (EEG), sleep EEG, 36 hours of ambulatory EEG recording, and brain-stem auditory evoked potentials were normal. Haematological tests were normal. There was a raised bone alkaline phosphatase activity at 142 IU (normal range 20–120 IU/l). Biochemistry was otherwise normal, including serum B12 and folate concentrations and thyroid function tests. Psychological testing showed full-scale IQ of 93 (WAIS-R), a verbal IQ of 96, and a performance IQ of 89 (pro-rated).

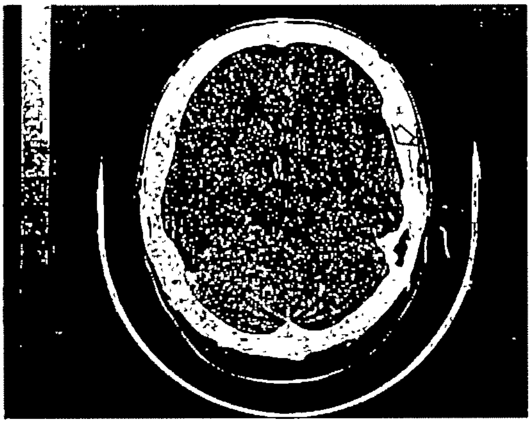

FIG. 1 CT scan showing decreased density in left temporal region and atrophy of the left temporal pole.

During a four-week trial off neuroleptic medication the patient became disturbed. He mumbled statistics to himself for hours at a time, urinated in inappropriate places, exhibited sexual disinhibition, but experienced no thought disorder, delusion or hallucination. He readily accepted small doses of chlorpromazine and within days resumed his previous lifestyle and pursuits, suggesting a sedative rather than antipsychotic effect.

Discussion

This patient demonstrated many of the clinical features commonly seen in Asperger's syndrome (Wing, 1981). He has a full command of English despite initial delay, an interest in astrology and numerology, a normal IQ, but suffers gross disturbance of social interaction and non-verbal communication. Wing (1981) also stresses lack of interest and pleasure in the first year of life. It is of interest to study such cases as the autistic process is not complicated by physical illness, mental impairment, or seizures, and in the case presented it seems reasonable to assume some role for the abnormal left temporal lobe in the genesis of the patient's mental condition.

As for the differential diagnosis, the patient was referred for assessment of possible temporal lobe epilepsy (TLE) or schizophrenia. Intensive EEG monitoring was normal and no attack characteristic of TLE or its auras was observed. During periods of apparent inaccessibility the patient was merely deep in thought, usually concerning statistics, and was always rousable. A diagnosis of schizophrenia seems untenable. Although he initially functioned well at school his overall illness seems continuous since childhood. He has never had auditory hallucinations, been deluded or thought disordered, and he has never had any classic negative features. In addition, his intellectual capacity for numerology, astrology, and astronomy is not only preserved but has become refined with time.

The diverse autistic syndromes almost certainly have multiple aetiologies. Several indirect lines of evidence have suggested temporal lobe pathology. Pneumoencephalograms performed on a heterogeneous group of autistic children showed enlarged temporal horns of the left lateral ventricles in 15 out of 17 studied. Five of the cases showed bilateral involvement (Hauser *et al*, 1975). Microscopic abnormalities in limbic structures have been demonstrated in a mentally impaired, epileptic autistic adult (Bauman & Kemper, 1985), but mental impairment and epilepsy in this study make interpretation difficult. De Long (1978) proposed a neuropsychological model of autism where a unilateral left medial temporal lesion coexists with an intact right side, a pattern seen in our patient's CT scan. This would result in impaired verbal learning and language function but normal processing of spatial information, as seen in autism.

It is well known that the incidence of obstetric complications is increased in autism (Gillberg & Gillberg, 1983) and that medial temporal lobe structures may be particularly susceptible to perinatal hypoxia (Brown & Brierley, 1973). Temporal lobe pathology has been demonstrated in several conditions having features in common with Asperger's syndrome, including TLE (Flor-Henry, 1969), schizophrenia (Slater *et al*, 1963), Korsakoff's psychosis (Hetzler & Griffen, 1981), and Kluver–Bucy syndrome (De Long, 1978). We present direct evidence of left temporal lobe pathology in a case of Asperger's syndrome. Although the neural mechanism involved cannot be elucidated, this case adds further weight to the notion of autism as a temporal lobe disorder.

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Organic Affective Psychosis Associated with the Routine Use of Non-prescription Cold Preparations

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A patient experienced an organic affective psychosis on three separate occasions after taking recommended doses of non-prescription cold/sinus preparations. The possible underlying pharmacological mechanisms of this clinical reaction lend support to the cholinergic–adrenergic balance hypothesis of affective disorders. Recognition of this acute drug-induced state can lead to appropriate short-term pharmacotherapy and can prevent misdiagnosis of a major affective disorder or schizophrenia.

Unusual or exaggerated responses to certain cold and sinus medicines have been reported. Hall *et al* (1985) reviewed the psychiatric reactions produced by decongestant drugs, including the sympathomimetics phenylpropanolamine, ephedrine, phenylephrine, pseudoephedrine, and naphzoline. Phenylpropanolamine, a common ingredient in decongestant and anorectic non-prescription preparations, has been associated with paranoia, seizures, and homicidal behaviour (Cornelius *et al*, 1984). Kane & Florenzano (1971) described paranoia, disorientation, and violence following the excessive ingestion of a mixture of hydroxyzine, ephedrine, and theophylline (Maraz). Herridge & A'Brook (1968) also reported ephedrine-related psychosis. Antihistamines have also been implicated in putative cases of drug-induced psychosis (Filloux, 1986; Climko, 1987; Wood *et al*, 1988). Thus cold and sinus medications have been associated with psychiatric side-effects, including confusion, paranoia, and affective disorder.

All of the reported cases of psychiatric syndromes associated with decongestant medications that we are aware of involved the overuse or abuse of these compounds. In this report, we present a longitudinal study of a man who had three separate admissions to hospital, with hypertension, paranoia, disorientation, and prominent affective symptoms, after taking the recommended dosage of sinus/cold medications. We believe that this is the first such case to be reported.

Case report

Mr A, a 49-year-old married farmer, was transferred from a local hospital to the medical service of our university hospital for further evaluation of hypertension and fixed, dilated pupils. On admission, he was found to be agitated, confused, paranoid, and delusional. His family reported that he had appeared to be in his usual state of health until he began taking a sinus medication containing phenylephrine and guaifenesin (Endal) two days before coming to the hospital. According to the patient's family, he was taking the medicine at the recommended dose (one to two tablets twice a day). Within a day after beginning this medicine, he developed paranoid ideation, along with decreased sleep and appetite, and the delusional belief that he had been choking his wife and would be punished for it.

The patient denied headaches, dizziness, chest pain, or any other symptoms. There was no prior history of hypertension or head injury. His wife stated that he had become agitated and confused about 13 years previously after taking "some type of sinus medication", and had been admitted to the same local hospital. Family history was negative for psychiatric or endocrine disease. On admission, his heart rate was 100 beats/min, respiration rate was 20/min, and blood pressure was 180/110 mmHg. Physical examination was unremarkable. He was an alert 49-year-old who looked somewhat older, with rapid and slurred speech. He was orientated to person and to place, but knew the year and month only. His affect was irritable, but he denied dysphoria. At times he appeared grandiose, believing that he had special abilities. His thought content was remarkable for the delusion that objects were sticking to his limbs because of his high blood pressure. He felt that he might suck up the objects in his room owing to this increased pressure, and believed that he needed to blow through his nose to reduce this risk. He knew the current president of the United States, but none before him. He correctly recalled four words immediately, but was unable to recall any of the words after five minutes. His digit span was five forwards and three backwards. His associations were loose and showed flight of ideas. He was unable to copy simple designs on paper, and his ability to perform abstract thinking was poor. Laboratory tests, electroencephalography, electrocardiography, chest X-ray, and computerised tomography of the head were all normal. A standard dexamethasone suppression test was negative. A neurology consultant found no lateralising features on physical examination. Cerebrospinal fluid studies, including bacterial and fungal cultures, were negative. Blood pressure remained moderately elevated (150/95–170/100 mmHg).

After several psychiatric consultations, the patient was transferred to the psychiatry service on the fifth day in hospital. Neuropsychological testing revealed a score on the Dementia Rating Scale of 108 out of a possible 144 points (normal range, greater than 140; Coblentz *et al*, 1973). Severe deficits in memory and conceptualisation were apparent, as was much perseveration. After two weeks in hospital, and treatment with thiothixene up to 10 mg per day, the patient was viewed by his family and his doctors to be at his baseline. He was no longer delusional, showed no cognitive deficits, and his sleep, appetite, affect, and blood pressure had improved. He was discharged with instructions to taper and then discontinue the thiothixene. The patient and his family were told that he should never take sinus or cold medications.

Approximately one year later Mr A was admitted to a rural psychiatric in-patient facility for evaluation of delirium, psychosis, hypertension, and dilated pupils. His wife stated that he had been having more sinus problems and had been taking an unknown non-prescription remedy. Two to three days after taking the medicine, he developed insomnia and decreased appetite. He began to speak in a rapid and disorganised fashion, and talked about a sensation of pressure in his head once again. No new psychosocial stressors could be ascertained from the patient or his family. It was discovered that he had been taking dexchlorpheniramine maleate (Polaramine), an antihistamine with anticholinergic properties, at a dose of 2 mg four times a day.

He was stabilised on low-dose haloperidol, propranolol, and nifedipine, and was released within two weeks to out-patient follow-up. On discharge he continued to take propranolol and nifedipine for his hypertension, which was now in the high-normal range. The haloperidol was gradually tapered and stopped three weeks after discharge. The patient and family were advised once again that he should never take sinus or cold remedies. Ten days after discharge he became dysphoric and talked of jumping into a pond. This reaction was short-lived, however, with mood normalising in another week.

Old records from the referring hospital confirmed that Mr A had been admitted to a psychiatric unit 13 years previously. Just before that admission he had been taking a cough/decongestant medication containing phenylpropanolamine, pheniramine maleate, pyrilamine maleate, dextromethorphan, terpine hydrate, and acetominophen (Tussagenic). He developed paranoid delusions, irritability, rapid speech, disorientation, auditory and visual hallucinations, and hypertension. He was treated successfully with low-dose haloperidol, with the later addition of imipramine. The discharge diagnosis was schizoaffective disorder, and no connection was made at that time between the episode and the cough/decongestant preparation.

Discussion

The patient described above was extremely sensitive to sympathomimetic/anticholinergic compounds. The use of certain cold/sinus preparations containing these substances was associated with the development of delirium and hypertension. This patient, like the others described in the literature, presented in an agitated, confused, and paranoid state. Unlike previously reported cases, he also exhibited prominent affective symptoms including irritable, dysphoric mood, and occasional grandiosity. Also, in addition to auditory and visual hallucinations, our patient experienced tactile hallucinations accompanying a delusion.

Recent literature has emphasised abuse of sympathomimetic amines and anticholinergic agents (Lambert, 1987; Whitehouse & Duncan, 1987; Climko *et al*, 1988), but there was no evidence of abuse of medications in the present case; the patient's family confirmed that he took the medications as prescribed, and they were dispensed to him by his wife. Further, his family was confident that he was not abusing alcohol or any other substances.

The cholinergic–adrenergic balance hypothesis of affective illness is consistent with both the phenomenology of this patient's illness and the associated anticholinergic and sympathomimetic effects of the medications. This imbalance hypothesis, first articulated by Janowsky *et al* (1972), relates the relative balance between acetylcholine and norepinephrine to mood disorders: a relative increase in central cholinergic tone can produce depression, while a relative increase in adrenergic tone can bring about mania. In the first episode described, the patient had been taking phenylephrine, which could produce adrenergic hyperactivity associated with agitation, hypertension, and dilated pupils. In the second episode, the anticholinergic effects of dexchlorpheniramine maleate may have reduced the cholinergic tone sufficiently to provoke manic symptoms and concurrent cognitive deficits. The later depressive phase could have been due to cholinergic rebound (Dilsaver, 1986) and/or the anti-adrenergic activity of the beta-blocker propranolol (Paykel *et al*, 1982). In the episode that had occurred 13 years earlier, phenylpropanolamine may have produced excessive adrenergic tone with a similar clinical presentation.

In conclusion, some individuals have rare exaggerated reactions to medications, including cold or sinus preparations containing sympathomimetic amines and anticholinergics or antihistamines. Lambert (1987) speculates that individuals with borderline personalities and affective disorders may be at greater risk for having a psychotic response to such preparations. These may be the same people who have increased sensitivity to accepted dosages of these medicines, or who may be at increased risk for abusing these medications. A careful review of all medications, including non-prescription medications, is indicated in all patients who present with acute psychotic symptoms or cognitive impairment. Recognition of the potential relationship between psychotic reactions and cold/sinus preparations can lead to more appropriate short-term management for those patients with acute drug-induced syndromes and may help to avoid a misdiagnosis of a major affective disorder, schizoaffective disorder, or a schizophreniform disorder.

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Coexistence of the Capgras and de Clérambault's Syndromes

J. M. O'DWYER

A patient with a paranoid schizophrenic illness showed features of two rare syndromes, Capgras and de Clérambault's.

Capgras and de Clérambault's syndromes are both psychiatric syndromes (Enoch & Trethowen, 1979). The literature contains few reports of the two syndromes occurring in the same patient (Simms & White, 1973). The Capgras syndrome is a rare and colourful syndrome, in which the patient believes that a person, usually closely related, has been replaced by an exact double. At the time of onset, there is often a feeling of ambivalence towards the subject of the delusion. de Clérambault's syndrome occurs when a patient, usually a woman, has the delusional belief that a man is in love with her. The 'victim' is usually older, and of much higher social status than the patient. He is initially usually unaware of the belief, and has done nothing to encourage such a notion. The patient usually acts on this belief.

The following is an account of a patient who, in the course of a paranoid schizophrenic illness, showed features of both syndromes.

Case report

ME, a 53-year-old single, unemployed, lady, was admitted in September 1988 under the provisions of the Mental Health Act 1983, because of her prominent delusions concerning her general practitioner, Dr X, whom she believed to be in love with her, but whom she also believed had been replaced by his exact double. This caused her a great deal of confusion and concern.

She was born locally, of English parents, and her early development apears to have been largely unremarkable. She never married, had had no boyfriends, and never had any long-term employment other than in hospital workshops.

The patient had a very long history of psychiatric illness. She first came to the attention of the psychiatric services at the age of 20, when she was diagnosed with paranoid schizophrenia. She spent 20 years in a long-stay ward of a psychiatric hospital, from where she was eventually rehabilitated and discharged into warden-controlled accommodation. There she managed poorly, frequently not eating, nor caring for herself, nor complying with her antipsychotic medication. This led to frequent deteriorations in her mental health, and numerous readmissions.

There was little information available regarding her family, with whom she had lost contact some years before. She has very few friends, and little social contact.

She has a 12-year history of diabetes mellitus, which is being controlled with oral hypoglycaemics. She has no other medical history, and no history of head injury. She does not drink alcohol or smoke. She denied any history of drug or substance abuse. She had had some involvement with the police, whom she frequently contacted to complain about apparent harassment by the neighbours. However, this was always a result of her delusional beliefs. She had never been charged or convicted of any criminal offences.

She was an old-looking, thin lady, who was otherwise quite well. Routine investigations were within normal limits, as were her blood glucose, electroencephalogram, and computerised tomography. She was rather dishevelled, tearful, and agitated. She was perplexed and bewildered on admission. Her mood was neither depressed nor elated. Her affect was inappropriate, with frequent outbursts of giggling while describing the disturbing events described below. Her speech was relevant and coherent.

She was deluded, believing that Dr X was in love with her. There was no evidence for this, he being happily married. She knew of his feelings, she claimed, because his wedding ring was twisted around on his finger. When asked about his wife, she claimed he was unhappily married, and would soon be leaving her. This she knew because he had recently lost weight. In response to this belief, she visited him at his surgery very regularly, and on occasion barged into his clinic, while he was examining other patients, and refused to leave. Between visits she telephoned him regularly, wrote letters, and sent him cards.

She claimed that on the night before her admission, her neighbour had lured Dr X into the flat next door. When he arrived four other men arrived too, who also went into the flat, where they attacked and killed Dr X, placing his body in the bath. Then, she claimed, they chopped his body into tiny bits, placed it in their car, and took it to Manchester Airport, from where it was to be taken to South America. She was to meet Dr X in South America, although she could not say how he would come to be alive there. She could not explain how she would get there, except to say that he would look after such details.

She claimed to have seen all of these acts being done, and gave vivid descriptions of those involved. She could not explain how that could be, as she had not left her own home during these events. She claimed to have heard the men talking and plotting, and described both second- and third-person auditory hallucinations, involving the four men, Dr X, and her neighbour. This mixture, in which it is difficult to disentangle the delusional and hallucinatory experiences (which usually occur in several modalities), has been described by Fish (Hamilton, 1986) as "fantastic hallucinosis". She had various auditory hallucinations, delusions, and visual hallucinations – the latter being of the extracampine type.

She was also convinced that, since then, Dr X had been replaced by an imposter, whom she described as being his exact double. She reported no difference between the two, except that the imposter did not smile as often. The reason that he had been replaced, she claimed, was an attempt to stop her leaving for South America, and meeting the real doctor, which she believed was the only way they could be happy. She persistently claimed that she was being forced to visit her GP by "other people", despite her best intentions not to. She was totally without insight. All of her cognitive functions were within normal limits.

Discussion

In this case, the Capgras delusion occurred after a 25-year history of paranoid schizophrenia. There was perplexity and bewilderment evident, as described previously by Davie & Freeman (1961). The personal specificity described by Enoch & Trethowen (1979) was also evident, the only person implicated being the GP. This is unusual, it being more common to have spouse, parent, or sibling involved; however, he was a key person in the patient's life at that time. The key mechanism behind the disorder is thought to be ambivalence, the patient loving and hating the subject simultaneously, and being unable to cope with the conflict. The hate is usually directed towards the new person, and is of recent onset. The patient, being unable to tolerate the ambivalence, splits the subject into good (old) and bad (new) persons, allowing hate to be revealed without any associated guilt.

The delusions regarding Dr X's love for her was earlier in onset than the Capgras delusion. According to Enoch & Trethowen (1979) there is an increase in affection and sexual craving towards the subject before the onset of the Capgras delusion. It is therefore perhaps surprising that the combination of syndromes does not occur more frequently.

Attempts have been made to describe the psychopathology of de Clérambault's syndrome, which is thought to revolve around the father–child relationship in early childhood years. There was not enough family history available to formulate an aetiological theory based on such relationships in this case.

The aetiology of both conditions in this case is closely related to the long-standing paranoid schizophrenic illness. There have been reports of Capgras syndrome occurring in a purely isolated form, and occurring subsequent to head injury

(Weston & Whitlock, 1971), with or without cognitive impairment. Erotomania is also described in a pure form, as well as occurring in paranoid schizophrenia.

Management of this patient has proved extremely difficult. Through the years she has been treated with varying types and doses of antipsychotic medication, all of which have had very limited success. She usually improves to some degree while in hospital, but when discharged defaults on her treatment, and rapidly relapses, leading to frequent readmissions. She has had both oral and depot medication. At the time of writing, she was a patient on a rehabilitation ward in hospital, and was being treated with trifluoperazine (15 mg t.d.s). The response was not spectacular, and she remained absolutely convinced of the apparent truth of her delusional beliefs, despite medication, which has been true of so many of her symptoms during her long illness.

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Dhat Syndrome
A Sex Neurosis of the Indian Subcontinent

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Dhat syndrome is a culture-bound sex neurosis of the Indian subcontinent. Fifty-two patients with a presenting complaint of passage of 'Dhat' in urine were studied. Diagnosis of neurotic depression, anxiety neurosis, hypochondriacal neurosis, and psychogenic impotence were made in 21, 19, 3, and 1 cases respectively. Seven patients received the diagnosis of pure Dhat syndrome, and one of gonorrhoea.

Dhat syndrome is a culture-bound neurosis quite common in the Indian subcontinent (Wig, 1984; Tiwari *et al*, 1986). Passage of a whitish discharge with urine is described as 'Dhat', believed to be semen by the patient, although there is no objective evidence of such discharge.

The word 'Dhat' has been derived from the Sanskrit word '*Dhatu*', meaning the elixir that constitutes the body. Seven types of '*Dhatus*' are described in *Sushruta Samhita*, an ancient treatise on Indian medicine. Semen is considered as the most important of these. Semen is known by the name '*Virya*' in India, derived from a Sanskrit word meaning bravery, power, or strength, or that which generates power and greatness. *Charak Samhita*, another ancient treatise on Indian medicine, describes a disorder resembling Dhat syndrome by the name of '*Shukrameha*' (spermaturia), in which the patient passes semen resembling urine or urine mixed with semen. In *Ayurveda*, the Indian system of medicine, loss of semen in any form is considered to lead to depletion of physical and mental energy. This belief is deeply ingrained in the Indian culture and is responsible for the symptoms of Dhat syndrome.

A typical case presents with multiple somatic complaints along with feelings of physical and mental exhaustion, attributed to the passage of semen in urine (Neki, 1973; Wig, 1984; Singh, 1985; Tiwari *et al*, 1986). Apart from a whitish discharge with urine there are no other urinary symptoms. Urine examination fails to reveal any discoloration, sperm, or any other abnormal constituents except for occasional oxaluria or phosphaturia.

Method

All patients seen in the psychiatry out-patient clinic with a presenting complaint of passage of 'Dhat' in urine over six months (January–June 1988) were included in the study. In addition to a detailed psychiatric assessment, patients were also asked what they thought caused the symptoms and how it had affected them. Urine examination was done in all cases for albumin, sugar, phosphates, and cytology.

Results

Fifty-two patients were included in the study. Most of them were in late adolescence or young adulthood. About half were married. Thirty-two patients belonged to families of manual workers. Others included farmers, shop owners, and clerical employees. Twenty patients were illiterate or educated to primary level. The rest had completed their schooling successfully, and five were graduates.

Twenty-eight patients were symptomatic for more than a year and four were ill for less than a month. Frequency of passage of Dhat varied from once or twice per day to once per 10–15 days, although in most patients it was once in 1-7 days.

More than 80% of patients complained of hypochondriacal symptoms (bodily weakness, multiple aches and pains, and vague somatic sensations). Depression was seen in half of the patients and 37 showed anxiety. Twenty-two complained of premature ejaculation and 19 of erectile impotence as a consequence of the passage of Dhat. Overconcern with nocturnal emissions was seen in ten cases. Urinary complaints in form of burning micturition and increased frequency were seen in five cases.

All the patients attributed their symptoms to the passage of Dhat and believed it to be semen. Twenty-four patients thought their earlier masturbatory habits to be responsible for the illness. Pre-marital and extramarital relations and earlier homosexual contacts were also believed to be the cause in 11 and 3 patients respectively.

Unexpectedly, none of the patients admitted any marital disharmony secondary to the illness, or sexual problems.

Urine examination was normal in most of the patients. Oxaluria and phosphaturia were seen in five and three patients respectively. Pus cells were seen in the urine of one patient who later received a diagnosis of gonorrhoea.

A diagnosis of neurotic depression was made in 21 patients. Another 19 were diagnosed with anxiety neurosis. Hypochondriacal neurosis and psychogenic impotence were the diagnoses in three and one case respectively. Only seven patients received a diagnosis of pure Dhat syndrome.

Discussion

Only 7 of our 52 patients received a diagnosis of pure Dhat syndrome. The rest could be given a diagnostic label of different neurotic illnesses except one, who on investigation was found to be suffering from gonorrhoea. The patients who received the diagnosis of pure Dhat syndrome did not differ from the rest in any significant way except for absence of anxiety, depression, or hypochondriacal symptoms. Whether these seven cases require a taxonomic niche of their own is a point which needs further investigation. It may be emphasised that in all the cases, symptoms were based on the cultural belief that loss of semen in urine was responsible for the problem.

In some of the earlier Indian studies (Nakra, 1971; Nakra *et al*, 1977; Singh, 1985), a large proportion of patients presenting with sexual dysfunction was found to be suffering from Dhat syndrome. In Nakra *et al*'s sample, semen loss was considered to be definitely harmful by two-thirds of sexual dysfunction patients and by 90% of patients with Dhat syndrome. Hypochondriacal symptoms, depression, and anxiety have been reported in varying proportions of Dhat syndrome patients in the literature (Sukhtankar, 1960; Joshi, 1965; Nakra, 1971; Nakra *et al*, 1977; Behere & Natraj, 1984; Singh, 1985).

There is an interesting Indian study (Malhotra & Wig, 1975) on attitudes in the general population towards loss of semen. A large proportion of the study sample believed it to be harmful, and this was more prevalent in lower socioeconomic groups. Malhotra & Wig (1975) comment that attitudes towards loss of semen constitute an organised belief system. The susceptible individual reacts to this system and presents with a variety of neurotic symptoms, which usually disappear if misconceptions about semen loss are effectively dealt with.

In some of the other reports from India (Carstairs, 1961; Singh, 1985) semen loss has been perceived as definitely harmful by large segments of normal populations studied. The belief was found to be more common in rural and less 'sophisticated' people.

There is a widespread cultural belief in India and neighbouring countries like Pakistan, Nepal, and Bangladesh, that semen is a very precious body fluid and its loss leads to serious physical and mental illnesses (Wig, 1984). *Charak Samhita* and *Sushruta Samhita* have described semen as an extremely precious material formed by distillation of blood. According to them, 40 meals give rise to one drop of blood and 40 drops of blood give rise to one drop of bone marrow. 40 drops of bone marrow give rise to 1 drop of semen, the elixir of life, one ejaculation of which will lead to loss of energy (Malhotra & Wig, 1975). Individuals, who are prone to neurotic problems may respond to such beliefs and present with Dhat syndrome as well as anxiety or depression.

In our experience such patients do not regularly attend for follow-up. A similar finding was reported by Singh (1955). A probable reason for this may be that these patients are not satisfied with our explanation that they are not suffering from any loss of semen and that semen loss is in any case not detrimental. Another possible reason may be that patients recover quickly. However, this point needs further investigation. In a study by Behere & Natraj (1984), 66% of the patients had completely recovered at one-year follow-up and another 22% had improved. Treatment modalities used were sexual counselling and treatment of associated anxiety, depression, or hypochondriasis. There are also reports of successful use of certain herbal medicine named in *Ashwagandha*, *Kapikachchu*, *Vriddhadaraka* and *Amrutprabha* in Dhat syndrome (Sukhtankar, 1960; Joshi, 1965). However, the results are controversial as sexual counselling and supportive psychotherapy were performed.

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Use of Carbamazepine in Psychosis after Neuroleptic Malignant Syndrome

MALCOLM PEET and JOHN COLLIER

A case is described of NMS during treatment with sulpiride. The subsequent psychosis resolved during treatment with carbamazepine. It is proposed that this patient may have suffered from a supersensitivity psychosis, and that resolution of her post-NMS psychosis could have been spontaneous

Patients who remain psychotic after recovery from neuroleptic malignant syndrome (NMS) pose a therapeutic dilemma. Rechallenge with neuroleptic drugs carries a significant risk of recurrence

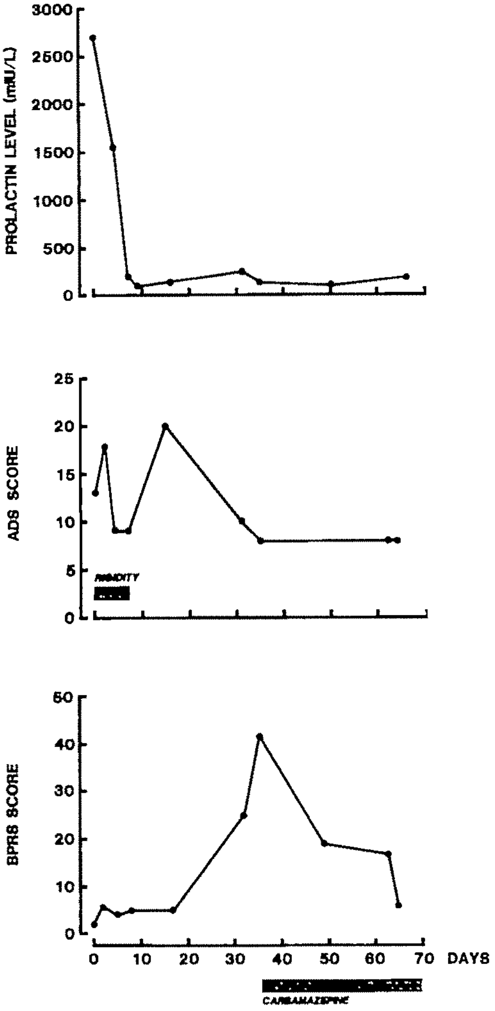

FIG. 1 Serum prolactin levels and scores on the Abbreviated Dyskinesia Rating Scale (ADS) and Brief Psychiatric Rating Scale (BPRS) after NMS (day 1 on chart is three days after its acute onset) and subsequent treatment with carbamazepine.

of this potentially fatal condition (Wells *et al*, 1988). Alternative treatments include lithium and electroconvulsive therapy (Shalev & Munitz, 1986).

We report on a patient who developed NMS while taking sulpiride, whose emergent psychosis after recovery from NMS resolved during treatment with carbamazepine. Psychopathology and movement disorder were quantified throughout the episode and serum prolactin levels were measured.

Case report

The patient is an unmarried 23-year-old woman of limited intelligence (formal IQ 75). She had been prescribed neuroleptics three years previously after a one-week history of appearing perplexed and uncommunicative, but with no definitive schizophrenic features. She had been treated sequentially with trifluoperazine, chlorpromazine, and thioridazine with some drug-free intervals during which she showed tardive dyskinesia (starting eight months after the initial prescription), and on one occasion, 20 months after the initial prescription of neuroleptics, a florid schizophreniform episode in which she was deluded and thought disordered.

She was first seen by us after the dose of thioridizine had been reduced and she had developed marked tardive dyskinesia. She begame agitated, fearful, and sleepless, and frequently stopped talking in mid-sentence. The thioridizine was discontinued and she was given sulpiride 200 mg twice daily which was increased to 400 mg twice daily three days later. Seven days after starting sulpiride she developed muscle rigidity, a reduced level of consciousness, sweating, tachycardia (110 per minute), tachypnoea (40 per minute), and hyperpyrexia (axillary temperature 38°C). Creatinine phosphokinase was 15 940 IU/l. She was treated with dantrolene to a total of 2 mg/kg.

Her subsequent clinical course is charted in Fig. 1. Ratings of dyskinesia were made on the Abbreviated Dyskinesia Rating Scale (Simpson *et al*, 1979), and psychopathology was rated on the Brief Psychiatric Rating Scale (Overall & Gorham, 1962). For the first week she remained very rigid, although this gradually reduced in severity. She was apyrexial during this time. Her prolactin levels were extremely high but fell during the week to normal. Tardive dyskinesia emerged and subsided without specific treatment. Her mental state then deteriorated. She was overactive, talkative, excited, suspicious, irritable, and hostile. She was deluded, believing that her valuables had been stolen, that she was a member of staff, and that her father was dead. There was no evidence of auditory hallucination or cognitive dysfunction. She was given diazepam (up to 8 mg q.d.s.) to control her agitation. Her psychosis worsened and she was prescribed carbamazepine in a dose increasing to 200 mg twice daily which gave a blood level of 4.8 mmol/l. Her mental state improved considerably within the first week of carbamazepine treatment and, after three weeks, her psychosis was resolved. She remains well on carbamazepine.

Discussion

Shalev & Munitz (1986), in a review of the world literature, found only five cases of NMS during sulpiride treatment, of which four had a fatal outcome.

This patient did not show convincing evidence of psychosis before the institution of neuroleptic treatment. She showed marked central nervous system effects of neuroleptic drugs, manifested by extrapyramidal side-effects and drowsiness, and the early development of tardive dyskinesia. Her first florid psychotic episode occurred only after prolonged neuroleptic treatment, and after tardive dyskinesia had already developed. It is therefore possible that this patient's acute psychotic symptoms were an expression of dopamine receptor supersensitivity consequent upon neuroleptic treatment. This concept of 'supersensitivity psychosis' was first put forward by Chouinard & Jones (1978) and Davis & Rosenberg (1979). This case has features in common with other reported cases of presumed supersensitivity psychosis occurring in non-schizophrenic patients (Caine *et al*, 1978; Sale & Kristall, 1978).

The acute psychosis subsided during treatment with carbamazepine, which is reported to be effective in hypomania (Cookson, 1987) and other excited psychotic states (Klein *et al*, 1984). Levenson & Fisher (1988) reported response to a combination of haloperidol and carbamazepine following NMS, but so far as we are aware, resolution with carbamazepine as a sole agent has not previously been reported. It is possible that the psychosis could have resolved spontaneously, independently of carbamazepine treatment. It is noteworthy that the tardive dyskinesia peaked and then subsided independently of any treatment, and it may be that the psychotic symptoms, particularly if they reflect a supersensitivity phenomenon, could form a 'hump' in the same manner, with eventual spontaneous resolution. This pattern was found in 4 out of 20 schizophrenic patients whose neuroleptic drugs were abruptly discontinued by Weinberger *et al* (1981) in their investigation of the supersensitivity hypothesis. Such speculations aside, it does appear that carbamazepine may be a useful alternative to lithium and electroconvulsive therapy in the treatment of post-NMS excited psychotic states.

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Incipient Neuroleptic Malignant Syndrome?

V. R. VELAMOOR, M. L. D. FERNANDO and P. WILLIAMSON

Recognising confusion as an antecedent to NMS may help abort the full-blown syndrome. Two such cases are reported, along with a third.

Neuroleptic malignant syndrome (NMS), a life-threatening complication of antipsychotic drugs, is often assumed to be rare. It is not clear whether it simply represents the extreme of the distribution of severity of extrapyramidal effects or whether it is actually a distinct entity. However, several observations suggest that rather than overestimating its frequency, we are more likely to have underestimated it (Pope *et al*, 1986). This may be due to the retrospective method of ascertainment, leading to cases, especially mild ones, going unrecognised. This observation is consistent with other reports (e.g. Destee *et al*, 1980).

The last four years have witnessed significant advances in the treatment of NMS. In addition to the obvious measure of prompt discontinuation of the neuroleptic, recent reports have shown that dantrolene sodium, L-dopa, and bromocriptine facilitate recovery, and are receiving increasing attention (Lazarus, 1986).

Estimates place the incidence of NMS between 0.2% and 1.4% (Lazarus, 1986). Early cases are rare in the literature, but if the episodes of 'milder' variants of NMS are included, the incidence may be as high as 12.2% (Addonizio *et al*, 1986). Changing diagnostic practices or detecting cases before the full-blown syndrome becomes manifest may arguably lead to a lower mortality. Although the onset of NMS has typically been described as abrupt and fulminant over 24–72 hours (Caroff, 1980), greater awareness and clinical suspicion may help abort cases in the very early hours of onset. This is consistent with earlier studies which describe partial forms of NMS preceding the onset of a full-blow syndrome (Goulon *et al*, 1983; Weinberg & Twerski, 1983.

Case reports

Case 1

A 41-year-old man was admitted with a diagnosis of manic–depressive illness. He was being treated with lithium but had discontinued the medication a couple of months earlier and had become hypomanic. He was excitable, restless with rapid speech and flight of ideas, and disorganised in his talk; he had lost 27 kg in weight in about 100 days. He received methotrimeprazine, a phenothiazine derivative (25 mg i.m. p.r.n.), lithium carbonate (600 mg b.i.d.), clonazepam (0.5 mg q.i.d.), haloperidol (10 mg i.m. every 4 h for 24–48 h), and benztropine (2 mg i.m. p.r.n.). The nurses reported that the patient had been voiding at will on the floor, smearing food over the walls, was very labile, and needed total supervision.

Upon admission he had been confused, drowsy, and restless. Because of this and a documented history of sensitivity to phenothiazines, investigations were ordered on an emergency basis as NMS was suspected. This suspicion was based on additional clinical findings of dehydration, warm and dry skin (temperature 35.4°C), and severe agitation. His creatinine phosphokinase (CPK) level at the time of admission to the general hospital was reported to be 5750 IU/l (normal range 38–174), serum sodium was 146, and anion gap was 16.8. Both aspartate (164) and alanine aminotransferase (71) levels were raised. Serum lactate dehydrogenase (LD) was 1347 (normal range 203–372) and the LD1 : LD2 ratio was raised to 0.83. The white cell count was 17.3, with an increase in granulocytes. He was treated in the intensive care unit with intravenous fluids, etc., and his CPK levels began to decline. The discharge diagnosis was NMS.

The patient returned to the care of his family physician on clonazepam (Rivotril) (1.5 mg t.i.d.) and diazepam (5 mg t.i.d.). No recurrence of symptoms of NMS or manic–depression have been reported since.

Case 2

A 60-year-old, retired man, with a diagnosis of manic–depressive illness and differential diagnosis of schizo-affective psychosis had been admitted because of increasing restlessness, agitation with pressure of speech, grandiose delusions, and elation of mood over two weeks. The patient had been receiving propranolol (10 mg b.i.d.) and trifluoperazine in progressively increasing dosage up to 10 mg q.i.d., discontinued after a few weeks in hospital. Chlorpromazine was also tried for a few days and discontinued. These medications were discontinued owing to severe extrapyramidal side-effects, as well as lack of efficacy. Other medications tried were clonazepam, diazepam, and methotrimeprazine, with lack of success. His psychosis remained unresolved and he began to appear confused, withdrawn, and depressed. His pulse, blood pressure, temperature, and respiration were monitored, and the CPK level was determined immediately (5750 IU/l, normal 38–174). Blood tests were repeated for assessment of isoenzymes. Total creatine kinase (CK) was 4524; CK3, 4524; total LD, 595 (normal 203–378); LD1, 17.4; LD2, 23.5 (normal 29–39); and LD3, 22.2. Both LD4 (16.1) and LD5 (20.8) were raised. The patient was suspected to be in the early stages of NMS and was referred to the local general hospital for management. With treatment, his CPK levels began to decline and other parameters returned to normal. Treatment was purely symptomatic and no neurological problems were reported. All medications, including diazepam, were discontinued and the patient was seen to make gradual and slow progress with resolution of psychosis as well.

However, the patient became psychotic with manic symptoms within a few weeks and was rechallenged with trifluoperazine (2 mg b.i.d.). As the symptoms did not subside carbamazepine (100 mg t.i.d.) was added to control manic behaviour a week later. This resulted in improvement in mental status and no signs or symptoms of NMS have been reported since. The patient is still in hospital.

Case 3

A 61-year-old man was admitted with a provisional diagnosis of catatonia, and haloperidol (5 mg t.i.d.) was prescribed. It was noted that he also had a tremor in his extremities and his tongue. On the tenth day of admission, he became extremely weak and he lowered himself to the floor, became stuporous, but not actually unconscious. He was described as being physically slow and very weak, such that he needed assistance to get on the examining table. He was also very pale and physical examination at that time revealed no rigidity. He was transferred to the general hospital where a tentative diagnosis of pulmonary embolus was made and he was started on heparin. This was not continued when he was transferred back to the psychiatric hospital as the diagnosis of pulmonary embolus had been disproven.

The patient continued to be mute with urinary incontinence. On the 25th day of admission he developed conjunctivitis in his left eye. An internist noticed spasticity in his extremities, an intermittent tremor of his right foot, sluggish deep-tendon reflexes, a slight ankle oedema (this was new), and petechial areas on the right calf. His leukopenia level was 3.7; there was an increased erythrocyte sedimentation rate (ESR) at 46, increase in liver function tests with a normal ammonia level, normal electrolytes, an increased creatinine level of 113, an increased blood urea nitrogen level of 11.1, a decreased albumin level of 39.2, and an increase in phosphorus levels. It was said that he had periods of agitation which would alternate with periods of extreme hypoactivity. He continued to be mute and negativistic. He was incontinent of both urine and faeces. His ankle oedema had progressed and he now suffered arterioventricular block. On perusal of his old chart, it became obvious that this was intermittent, with or without superventricular tachycardia. This, therefore, seemed to be a problem independent of his suspected NMS.

On the 38th day of admission, the patient was transferred to the general hospital. He arrived in hospital in a catatonic state. He had cogwheeling and a general rigidity. His temperature was elevated at 37.5°C and his heart rate was elevated at 100 beats per minute. His liver function tests were elevated and his CPK was 999. He was placed on 5 mg diazepam every six hours p.r.n.

On the second day of admission to the general hospital the patient suddenly worsened. His temperature rose, he became increasingly rigid and unresponsive, and his blood pressure increased. His CPK rose to 3589. There was an increase in his chloride and sodium levels, a decrease in his albumin, an increase in his aspartate aminotransferase (290) and levels of D (390), a creatinine level of 83, and normal arterial blood gases. Interestingly, he showed no signs of leukocytosis. His electrocardiogram was normal. A nephrologist was consulted and he was started on bicarbonate-saline diuresis. Computerised tomography of his head revealed no abnormality. His condition improved significantly over the subsequent 12 to 24 hours. On the 10th day he returned to the psychiatric hospital. His CPK levels dropped to 63.9.

By about the third month of admission to the psychiatric facility, the patient was described as continuing to be withdrawn, agitated, and mute. As the patient was described as being assaultive, at times, to other patients, he was started on thioridazine and diazepam, but because of the decrease in white cell count, these were discontinued. However, they were recommenced owing to his agitated and at times aggressive behaviour, and his white cell count has increased to normal limits and his CPK levels, which are being monitored on an ongoing basis, have been within the normal range. He has been maintained on thioridazine (20 mg t.i.d.) as any further increase in dosage resulted in elevation of CPK levels. The patient is still in hospital.

Discussion

The three cases described here ostensibly lend credence to the possibility of what one might call incipient NMS. Case 1 was afrebrile throughout, although he had marked rigidity and tremors. Apart from the isolated finding of an elevated CPK level and leukocytosis, there was no abnormality in investigations. Adityanjee *et al* (1988) are of the view that NMS cannot be diagnosed in the absence of even one of the four essential clinical components, namely, altered sensorium, muscular rigidity, hyperpyrexia of unknown origin, and autonomic dysfunction. Only altered sensorium (confusion) and muscular rigidity were present in Case 1. However, elevation of the serum CPK level to 5750, associated with leukocytosis, can be considered as supportive features. The authors are in agreement with Adityanjee *et al* (1988) that undue reliance may have been placed on elevated CPK levels as a diagnostic parameter. However, we would differ with them that firm evidence for considering the syndrome as an extension of extrapyramidal symptoms is lacking. This belief probably stems from their rather stringent four criteria.

Case reports of early recognition and prompt management have mushroomed in recent years and estimates of its prevalence have suggested that although it is not common, NMS is not rare. There is not sufficient evidence yet to refute the 'spectrum concept'. Clinical differentiation between lethal catatonia and NMS can also be difficult. Published case reports of the two syndromes indicate differences in mode of onset, signs and symptoms, and outcome (Castillo *et al*, 1989). While lethal catatonia often requires neuroleptic treatment, NMS necessitates immediate cessation of neuroleptics. The borderland between NMS and lethal catatonia remains to be fully explored.

In Case 2, again, confusion and muscular rigidity (essential clinical parameters) associated with CPK levels of 1625 IU/l as supportive feature raises the possibility of early NMS, which may warrant a label of incipient NMS.

In Case 3, all four essential components were present. This was associated with an increase in CPK levels and not leukocytosis but leukopenia, perhaps arising through his chronic neuroleptic usage. The medical complications as a consequence of cardiac rhythm problems also seems to have preceded the onset of NMS.

In Cases 1 and 2, the main clinical feature seems to have been confusion resulting in a suspicion of NMS. This is an important finding and needs to be examined further. It may be possible to pick up and abort early cases (incipient NMS) if there is a higher index of suspicion with regard to this particular parameter. Van Putten *et al* (1988) reported a patient with NMS manifesting as a prolonged confusional state. They speculated that their case was a mild one, presenting primarily with confusion, and mild elevation of temperature and blood pressure. If so, such milder cases are probably not all that infrequent. Furthermore, they reported that there was less confusion with 5 mg than with 10 mg fluphenazine. This accords with previous studies (Shalev & Munitz, 1986; Gelenberg *et al*, 1988) which focused on dosage and the tendency of neuroleptics in higher dosage to predispose to a greater likelihood of developing NMS. Confusion as an important antecedent to the full-blown syndrome appears to be more prevalent with drug combinations, especially those involving lithium (Prakash *et al*, 1982; Cohen & Cohen, 1974).

In Case 3, the diagnosis of NMS was made in the absence of leukocytosis. Stopping the neuroleptics during the initial phase possibly aborted the full-blown episode of NMS, as probably happened in Cases 1 and 2.

NMS occurs very rapidly, and in more than 90% of the cases the full syndrome may be present within 48 hours of the first symptoms (Shalev & Munitz, 1986). Our reports show that early detection and active treatment is associated with a much lower mortality. In any event, a patient developing NMS should be moved immediately into medical intensive care and supportive measures should be used. By discontinuing the neuroleptic and initiating prompt therapy, symptom resolution in NMS normally occurs within ten days (longer for depot neuroloptics). A correlation may be found between recovery from the syndrome and the decline in urinary metabolites of the neuroleptic (Allan & White, 1972; Hashimoto *et al*, 1984).

Caution must be used during rechallenge of patients who have had NMS. The rechallenge should be avoided if possible until after the syndrome has resolved. Our focus on low dosage, picking up early signs (e.g. confusion) and greater vigilance on the part of physicians as well as nursing staff in aborting incipient cases may bring about greater understanding and effective management of these patients. This approach will also provide clues to further research in this area.

Acknowledgements

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British Journal of Psychiatry (1990), **156**, 585–595

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Editor: Ian Pullen

Contents: The ethics of prophylactic trials of depression/Prison suicides/Outcome in unipolar affective disorder after stereotactic tractotomy/ Forensic aspects of mental handicap/Asian patients and the HAD scale/Physical health and unmet needs/ Was Hitler a Christian?/Intestinal permeability in schizophrenia/'New long-stay' patients and social deprivation indices/Platelet MAO and 5-HT uptake in agoraphobics/'Le Suicide'/There are none so double-blind . . ./Malaria presenting as atypical depression.

The ethics of prophylactic trials of depression

SIR: For over twenty years psychiatrists have been aware of the importance of continuation therapy with antidepressive agents for prevention of relapse of unipolar depressive illness. Over a dozen studies of continuation therapy have shown a significant advantage for antidepressants over placebo. Montgomery *et al* (1988), dissatisfied with poor definition of relapse versus recurrence, have now reported a multicentre prophylactic study which seems better controlled and executed than any other to date. They have shown that patients who had been recovering for four months from major unipolar illness had a lower relapse rate (20%) continuing for a year on fluoxetine than a control group (54%) maintained for the same period on placebo. Fluoxetine, a relatively new drug, is the only one studied in this way so far. However, even before Dr Montgomery *et al*'s work it was apparent from the literature that the risk of relapse in the first year after recovery amounted to about 50% of placebo users and 20% in those who seemed to have kept up their medication. The serious implications of relapse for the lives of patients are apparent. Despite this knowledge, depression is notoriously badly managed in medical and, possibly, in psychiatric practice. This may be because many patients will not follow good advice or (perish the thought) because good advice is not given.

From Dr Montgomery *et al*'s own review of literature it would appear that no well-controlled study of prophylaxis or continuation has failed to show superiority of drug over placebo, irrespective of the type of antidepressant studied. It follows that subjects recruited into prophylactic trials of the Montgomery type (difficult and lengthy undertakings) should be advised that they are being asked to accept a very high risk of depressive morbidity if they consent to participate. The extent of this risk is difficult to estimate since no published accounts are available on the outcome of placebo relapsed depressives with renewed treatment. Furthermore, the fate of drop-outs is seldom fully reported in follow-up studies. It is noteworthy that the exact wording of 'informed consent' forms is seldom reproduced in the text of published papers. This poses the question of whether subjects of trials such as that of Dr Montgomery *et al* really understand the extent of risk that they are being asked to undergo.

Is the distinction between relapse of the original episode and true recurrence after recovery (insisted upon by Dr Montgomery *et al*) of sufficient importance to justify one year placebo-controlled trials? To the patients, the distinction may appear as mere academic hair-splitting. We already know that these illnesses get worse, not better, over time and that relapse rates rise throughout life. Every psychiatrist should reflect on whether he/she would encourage his/her close relatives to be enrolled in prophylactic studies of the type described. It is conceivable that many will think (as I do) that the price to be paid for more prophylactic trials of classic, placebo-controlled design is unacceptable.

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MONTGOMERY, S. A., DUFOUR, H., BRION, S., *et al* (1988) The prophylactic efficacy of fluoxetine in unipolar depression *British Journal of Psychiatry*, **153** (suppl. 3), 69–76

SIR: There is consistent support from many placebo-controlled studies that in the period following recovery on an antidepressant, further treatment is needed to consolidate the response. An analysis of the relapse rates on placebo of first-time depressives who would not normally be expected to develop a new episode of depression for some time shows a very similar relapse rate compared with that seen in recurrent depressives (Mindham *et al*, 1973). This strongly suggests that for those who stop antidepressants immediately after response, relapse in the subsequent six months is a return of the symptoms of the inadequately treated depression. It is necessary to continue treatment for at least four months following response to ensure that the episode of depression is properly treated. This simple advice is often ignored, sometimes by patients but mainly, we believe, by doctors, who seem as yet to be unaware of the importance of this. The average duration of treatment for a course of antidepressants in the UK is currently estimated to be six weeks.

The study of fluoxetine (Montgomery *et al*, 1988), which Professor Fahy praises did not address the issue of maintenance treatment to consolidate the response. All patients who consented to the study were treated for a six-month period with the antidepressant to ensure a persistent response. This study was attempting to address a different issue: whether long-term treatment in recurrent unipolar depression reduced the chances of the development of further new episodes of depression. This is not as Professor Fahy states, mere hair-splitting. The use of a drug to treat an episode of depression is quite different from the use of a drug to prevent a new episode of depression arising, a distinction emphasised by Montgomery (1989). There are very few good studies for secondary prophylaxis although a case can be made for fluoxetine, imipramine and possibly lithium. With amitriptyline, design flaws in the two small studies conducted leave room for doubt For most antidepressants, we do not know whether they are effective in prophylaxis or not.

The point where we take issue with Professor Fahy is his assumption that any antidepressant will do as they all, he implies, have efficacy in maintenance and prophylactic use. He appears to find it ethical to offer long-term treatment with an antidepressant although there are doubts about long-term efficacy. We would prefer to be open with our patients and tell them that some like Professor Fahy believe that any antidepressant works while others like ourselves have doubts. We would invite the patients to participate in a carefully-controlled study to help establish this important point. We would spell out the risks and benefits in order to help them make an informed decision. This seems to be the most ethical course to adopt.

Much of Professor Fahy's belief that any drug will do is based on his understanding that no well controlled investigation of continuation or prophylaxis has failed to show efficacy. This approach is risky. Lithium was found to be no better than placebo in recurrent unipolar depression by Prien *et al* (1984). More recently, Georgotas *et al* (1989) reported that nortriptyline was ineffective compared with placebo whereas phenelzine was effective in a one year study of prophylactic treatment. Results like these emphasise our need for caution and suggest that we should require a convincing demonstration of long-term efficacy of an antidepressant, preferably against placebo, to avoid putting patients at risk.

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Prison suicides

SIR: Dooley (*Journal*, January 1990, **156**, 40–45) draws attention to the important problem of suicide in prison, but makes some inferences from the data which are questionable, and comes to conclusions which are disappointing in making so few recommendations for change.

Firstly, is it true that suicide in prison is increasing? The rates quoted by Dr Dooley for 1976–1987 are remarkably similar to those given by Topp (1979) for 1958–1971. It may be that in the years 1972–1975 there were relatively few suicides and that the rate has simply returned to baseline. The general characteristics of prison suicide described by the two authors are very similar, suggesting a stable rather than a changing phenomenon.

Secondly, how good is the evidence that prison suicides are a consequence of mental disorder? The prevalence of past psychiatric history quoted is 33%, again very similar to that of Topp (38%) and Backett (39%) (1987) These rates are no higher than have been found among general hospital patients admitted after episodes of non-fatal deliberate self-harm, the majority of whom are socially deprived and distressed rather than mentally ill. They are lower than those found among people who kill themselves outside prison, the majority of whom may be mentally ill. In this respect the sentenced prisoners described by Gunn *et al* (1978) are not an adequate control group, since half of Dr Dooley's sample were on remand or unsentenced.

Common sense might suggest that people who kill themselves in prison do so *because* they are in prison. The data tend to support that view, since suicide has been such a persistent risk of imprisonment across the years and is not overwhelmingly associated with mental illness. Again, surely the explanation for an increase in prison suicides in the summer months is that it is during those long hot days that loss of liberty becomes hardest to bear?

Such an interpretation leads to the conclusion that it is institutional reforms which are needed if the suicide rate in prison is to be reduced, and two spring readily to mind. Firstly, fewer people should be held in prison on remand. The proportion of non-sentenced prisoners in Topp's cohort was 37%; in Dr Dooley's it was 47%. Among the 68 people who killed themselves in prison in 1987 and 1988, 74% were on remand (*Hansard,* 1989). These figures confirm that remand prisons are unsuitable places in which to assess and respond to the psychological and social needs of vulnerable and distressed people (Howard League, 1986, 1989) Secondly, we should make far less use of custodial sentencing for non-violent crimes (Blom-Cooper, 1988). Half of Dr Dooley's sample killed themselves while imprisoned for crimes against property. We no longer hang people for murder; it is a great irony that they should hang themselves while in prison for lesser offences. Conditions in British prisons are a national scandal: they are overcrowded, disorganised and insanitary. If they were emptied of trivial offenders it would be for the general good. It would also increase the opportunities for a reformed prison medical service to respond to the needs of the long-term prisoner whose suicidal despair is (as Dooley points out) so often tragically unidentified under the present system.

In the 1890s the authorities responded to the problem of suicide in prison by putting up safety netting between landings. In the 1990s, should they respond by redesigning cells so that desperate prisoners cannot garrotte themselves on their bars? We must do better than that. The suicide rate in prisons raises important ethical questions about our social and penal policies. Psychiatrists (both individually and through the College) should press for reforms in the prison system with a vigour equal to that which they show in proposing reforms in the practice of forensic psychiatry in other countries. To do less is simply hypocritical.

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Outcome in unipolar affective disorder after stereotactic tractotomy

SIR: The report by Lovett *et al* (*Journal,* October 1989, **155,** 547–550) confirms our finding that a better outcome, especially in unipolar affective disorder, can be achieved by simpler surgical techniques such as bimedial frontal leucotomy (Hussain *et al,* 1988).

In our series, there were 15 cases of unipolar affective disorder. Of the 13 who had a non-stereotactic operation, 11 had a good outcome at follow-up, and of these, eight achieved a complete recovery. Neither of the two stereotactic cases however, had a good outcome. Our study was similar to Dr Lovett's in, for instance, the long histories of depressive disturbances in mood (either recurrent or continuous), the resistance to all forms of treatment available, and the assessment approach. However, the length of follow-up was longer in our study – a mean length of 108 months, compared with 69. It would seem that the stereotactic tractotomy lesion in the lower medial quadrant of the frontal lobe is unable to give a better result than the simpler modified bimedial approach, which not only produces a lesion in the lower medial quadrant but also severs the connection from the frontal lobe to the cingulate gyrus – the fronto-limbic northern pathway (Kelly, 1976).

There has thus been support for this finding and for providing this simpler form of surgery, which can be done at a regional level (Snaith, 1989).

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Snaith, P (1989) Is psychosurgery yesterday's treatment? *British Journal of Hospital Medicine*, **42**, 13–14

Sir: In their recent paper, Lovett *et al* (*Journal*, October 1989, **155**, 547–550) repeat the advice from a previous paper (Lovett & Shaw, 1987) that stereotactic tractotomy may give a poorer outcome in patients with organic cerebral changes. They again quote a bipolar case (no. 4) from their 1987 series as showing a particularly poor course after, and possibly before, the woman's second operation in 1981 This was associated with embolic cerebral damage.

Unknown to the authors, this woman, two-and-a-half years after her second operation, showed an almost complete resolution of affective symptoms that allowed her eventually to be transferred from long-stay to out-patient care. For the last five-and-a-half years she has suffered from neither mania nor depression, and her only psychotropics are diazepam (5 mg) in the morning and temazepam (20 mg) at night. If anything, her organic cerebral changes came on after the extension operation, since a preoperative Emergency Medical Information scan was reported as normal. Since that time she has experienced several cerebral infarctions in both frontal areas from emboli due to mitral valve disease, but has recovered well from the resulting brief hemiplegia and dysphasia.

She walks normally, although her speech can become muddled when she is under pressure, and very occasionally she is a little 'high' and 'interfering' for some hours at a time.

I have no doubt that this woman would never have been discharged nor have been free of affective illness without tractotomy. The recovery two-and-a-half years after the operation may seem late, but it may possibly have been delayed by the stroke eighteen months after operation, or alternatively the stroke may have represented a further and curative extension of the limited surgery to her frontal tracts.

Certainly, it would seem too early to regard cerebral changes as an absolute contraindication to psychosurgery

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Forensic aspects of mental handicap

Sir: Turk's annotation on the forensic aspects of mental handicap (*Journal*, November 1989, **155**, 591–594) is timely in bringing this issue to prominence. However, I feel that he omits to mention an important cause of distortion in the available figures relating to mental handicap and criminal offences, namely the great reluctance of the police to prosecute people with a mental handicap. As a result, the available figures on any relationship which may exist between crime and mental handicap are not a true reflection of the situation. For example, over the last six months I have seen six individuals each of whom have committed what would normally be regarded as criminal offences, including assault, theft, destruction of property, sexual offences, and various minor nuisance offences. None of these people were prosecuted by the police although they were, in three cases, brought into our hospital by the police. It is of course debatable how far prosecuting an individual with a mental handicap serves any useful purpose, and this is not a plea for the wholesale prosecution and imprisonment of all mentally handicapped offenders. However, until we can collect more accurate figures about offences committed by people with mental handicap, it is misleading to use existing figures on convicted persons as an indication of the presence or absence of relationships between intellectual retardation and criminal behaviour.

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Asian patients and the HAD scale

Sir: I was puzzled by the letter from Chaturvedi (*Journal*, January 1990, **156**, 133) as it appears he has failed to grasp the content of my article (*Journal*, October 1989, **155**, 545–547). My article was not about a 'mere translation' of a scale for use with Asian psychiatric patients, but a study to validate a translated version of the Hospital Anxiety Depression (HAD) Scale against the Standardised Psychiatric Interview. In fact, I came to the conclusion that 'in its present form the translated version of the HAD scale has a limited role as a screening instrument', and I have stated that I do not recommend its use for the diagnostic purpose.

The myth of depression in Asian patients with its different presentation is not valid any more. The World Health Organisation (WHO) study (1983) is one of many which suggest that the core symptoms of depression are similar in different cultures. With appropriate examination, one can elicit psychological symptoms of mood disorder in patients from Asian or any other culture. As my studies suggested, somatic symptoms did not correlate with depression, not only on the HAD Scale but also on the Clinical Interview Schedule (CIS) (this interview has been used previously in many cultures by different authors).

I fully agree with Dr Chaturvedi that without proper validation an instrument should not be used in a different culture. In fact, my study supports the notion that to avoid the western bias the psychiatric instrument should be built from scratch in that very culture (Leff, 1988). While such works (as I know from personal contact) are being carried out in other countries, the modified form of instruments from western cultures have a role in bridging the gap. The cross-cultural studies are important for better understanding of mental illness; this type of work will help by giving a starting point from which to carry out further research.

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SIR: Chaturvedi's (*Journal*, January 1990, **156**, 133) criticism of the use of the HAD Scale with Asian patients is too sweeping although it is true that Nayani's evaluation of his own Urdu translation of the HAD Scale (*Journal*, October 1989, **155**, 545–547) is incomplete and that previous cross-cultural research in psychiatry has often ignored issues of cultural and conceptual validity.

Together with colleagues in Lahore, Pakistan, I have recently completed an evaluation of a new translation of the HAD Scale in Urdu (Mumford *et al*, 1989). We used a five-stage process: (a) initial drafting in Urdu by six independent translators; (b) translations back into English and modification of the Urdu version; (c) evaluation of linguistic equivalence of items in a large bilingual population; (d) evaluation of conceptual equivalence by examining item-subscale correlations; and (e) evaluation of scale equivalence by two-way classification of high and low scorers.

Our results do not justify the scepticism of Dr Chaturvedi, but suggest that the HAD Scale may be a useful and reliable instrument for use in Pakistan. Further validation studies in clinical populations are in progress.

The eventual aim is of course to develop new psychological tests for use in Pakistan. However, the translation of selected western instruments into non-western languages can be a useful interim measure When properly evaluated and validated, these do have the advantage of allowing some cross-cultural comparisons to be made.

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Physical health and unmet needs

SIR: Brugha *et al*'s paper (*Journal*, December 1989, **155**, 777–781) on medical needs of the chronically mentally ill in community care recommends close medical supervision and regular checks on physical health.

If one considers the high co-morbidity of medical and psychiatric disease, we agree that regular attention to physical health should be given to this vulnerable population. However, the way in which this attention should be given remains unclear from their paper. Surely the authors cannot justify regular laboratory tests on the basis of their data. Of their sample of 145 patients, only 12 patients had unmet medical needs (8%) requiring further medical investigation (10 cases) or treatment (2 cases). These data are in line with findings of Maricle *et al* (1987) and Honig *et al* (1989).

Unmet medical needs can often be dealt with by the patients' family doctor (Maricle *et al* 1987, Honig *et al* 1989). A well-informed general practitioner (GP) can perform an interview followed by a physical examination, and if necessary, specific laboratory tests. However, the GP needs a lot of support to be able to do this properly in the chronic psychiatric population.

A more feasible approach therefore for any community mental-health-care worker is to alert oneself to possible signs and symptoms of physical disease whenever a patient is seen. When in doubt, the supervising community psychiatrist or the patient's GP should be consulted. If necessary, a direct contact between the patient and the GP should be established even if this would mean accompanying the patient to the GP's surgery and explaining the problems. This procedure would promote the reintegration of the patient back into the appropriate ambulatory medical service. A close co-operation between the community mental health service and the patient's GP would be more effective, of higher medical quality (clinical assessment, physical examination and specific laboratory tests) and less expensive than the suggested routine checks in the day-hospital setting. This physical screening procedure is satisfactorily in use in our Community Psychiatric Unit.

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Was Hitler a Christian?

Sir: I found myself agreeing with the central anti-racist thrust of Samuel's letter (*Journal*, October 1989, **155**, 568–569) but cannot let pass his extraordinary assertion that "Stalin, Hitler, Mussolini, Franco and Rudolph Hoess were all Christians". The Shorter Oxford Dictionary defines a Christian as a "person believing in, professing or practising the religion of Christ ... a person showing character consistent with Christ's teaching". My nodding aquaintance with 20th century history suggests that none of these figures fit even this wide and non-denominational definition. Stalin was actively anti-Christian while Hitler, Mussolini and Franco saw the Church as a useful institution to be manipulated for political ends, nothing more. I know no details of Herr Hoess' theological views.

Dr Samuel's letter perhaps reveals a common misuse of the word 'Christian' as a synonym for 'Western'. It is inaccurate, misleading and potentially offensive to both Christians and non-Christians.

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Intestinal permeability in schizophrenia

Sir: Lambert *et al* (*Journal*, November 1989, **155**, 619–622) recently reported that the intestinal permeability of schizophrenic patients, determined by urinary excretion of ingested ^{51}Cr EDTA, was not significantly different from that of normal subjects. They concluded, "schizophrenia is, at least in the majority of cases, unrelated to coeliac disease" since the latter shows a highly significant increase in intestinal permeability to ^{51}Cr EDTA.

These authors imply their results are relevant to my hypothesis of the genetic relationship between the two diseases (Dohan, 1988). They overlook the possibility that *some but not all genes* necessary for susceptibility to coeliac disease are also present in those hereditarily susceptible to idiopathic schizophrenia. This possibility was suggested by clinical observations indicating the two diseases occurred in about 2–5% of patients with a primary diagnosis of either disease – at least 10 times as frequently as chance expectancy. As expected from the co-occurrence of the two diseases noted above, two of the 24 schizophrenic patients (8%) studied by these authors exhibited intestinal permeability well within the coeliac disease range. However, no diagnostic studies for coeliac disease were mentioned.

I have hypothesised (Dohan, 1988) that abnormal alleles in both diseases code for enhanced gut-cell receptor activity for the glutamine-rich gluten peptides and that aberrant alleles at two or three loci coding for defective systemic enzymes catabolising gluten peptides are the same in coeliac disease and idiopathic schizophrenia. In addition, I postulate idiopathic schizophrenia also requires a schizophrenia-specific gene. This I suspect causes brain dysfunction because of preferential binding of opioid peptides, exorphins, derived from glutens

(Zioudrou *et al*, 1979), to certain brain receptors which normally modulate dopaminergic and cholinergic activity.

Coeliac disease requires abnormal alleles at another locus. This probably codes for increased immunological response to gluten antigens and is in linkage disequilibrium with certain human leucocyte antigen (HLA) loci. These loci occur in most coeliac patients but are as low in frequency in schizophrenics as they are in the general population. Thus most patients with idiopathic schizophrenia do not have coeliac disease, but a small per cent do because they have all the genes for both diseases.

A previous article (Dohan, 1988) presents the evidence for and against my hypotheses. In addition to dietary trials it includes: the extreme rarity of schizophrenia in populations consuming little or no grain; the exceptionally high correlation between rates of wheat and rye consumption and first admissions to mental hospitals of women with schizophrenia ($r = 0.98$, $P < 0.001$); the increased excretion of small peptides, some with opioid activity, by schizophrenic patients (Reichelt *et al*, 1985); the stereotyped behaviours and patterned seizures in rats, hours after intracranial injections of gluten peptides; the changes in schizophrenia-relevant neurotransmitters in cats' brains produced by chronic high-gluten ingestion (Thibault *et al*, 1988).

My article suggests many ways of testing my hypotheses. For example, Bruce *et al* (1985) reported increased transglutaminase activity in gut biopsies of active and remitted coeliac patients which might be important in gliadin binding to gut tissues. This, I postulated, might increase the transcellular passage of glutamine-rich peptides, including exorphin precursors, across the gut barrier. What is the activity of this enzyme/receptor in schizophrenic patients?

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Sir: Abnormal intestinal absorption has been suggested to be an aetiological factor in schizophrenia. In an earlier study, the cellobiose/mannitol test, which is a reliable index of intestinal permeability, was carried out on long-stay psychiatric in-patients (Wood *et al*, 1987). Each patient was investigated by duodenal biopsy for the presence of abnormalities of the mucosal morphology of the small intestine. The study showed that 34% of the patients had abnormal absorption which could not be attributed to established bowel disease. Patients who were receiving neuroleptic medication but not anticholinergic drugs were those who most frequently showed abnormal intestinal permeability. These findings were considered to suggest that a proportion of patients with schizophrenia have abnormal gut permeability which might result in increased absorption of molecules which induce psychosis, but which at the same time, protect against the development of Parkinsonian side effects of treatment.

Following these earlier findings, we set out to test the hypothesis that within an otherwise unselected group of chronic schizophrenic patients there are two populations, one taking neuroleptic medication without anticholinergic medication and showing abnormal intestinal permeability, and another taking both neuroleptic and anticholinergic medication but showing normal intestinal permeability. The cellobiose/mannitol test was used in two groups of in-patients with schizophrenia (Research Diagnostic Criteria (RDC); Feighner *et al*, 1972), both of which were receiving neuroleptic drugs but only one of which required anticholinergic drugs. It was difficult to find in-patients fulfilling RDC criteria for schizophrenia who were not taking anticholinergic medication and who could give informed consent. Consequently, only 25 patients were recruited, 16 receiving anticholinergic drugs and 9 not receiving them: we had hoped for larger, more balanced groups. Eleven patients (44%) had abnormal intestinal permeability, but they were evenly divided between the groups. This rejected our hypothesis, albeit in a very small sample.

Five patients with normal tests in Wood *et al*'s (1987) study were re-tested and three had abnormal results. We can offer no satisfactory explanation for this finding. The cellobiose/mannitol test is not significantly affected by extraneous factors such as gastrointestinal transit times and renal function, and the results cannot be explained by changes in the patients' medication. The fact that three patients with chronic schizophrenia developed abnormal intestinal permeability over time argues powerfully against the latter's role as an aetiological factor. The questions remain as to what the mechanism of abnormal intestinal permeability is, and what influence it has on both the disease process and the efficacy of medication.

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'New long-stay' patients and social deprivation

Sir: Abbott (*Journal*, January 1990, **156**, 133) makes the point that a detailed analysis of the socio-demographic characteristics of catchment areas may reveal associations between accumulation rates of 'new long-stay' patients (admission duration of more than one year) and indices of social deprivation. At present, the Team for the Assessment of Psychiatric Services (TAPS) is evaluating the reprovision of services for patients leaving Friern and Claybury Hospitals. Furthermore, such an analysis has already been performed (Jones & Margolius, 1989) in which the Spearman rank correlation coefficient between the annual rate of accumulation of new long-stay patients and the Jarman-8 index (Jarman, 1983) of social deprivation for their nine districts of residence was 0.82 ($P<0.01$).

Although this association has not been previously reported, it is not surprising. The association between residential area and incidence of schizophrenia was described in Chicago over 50 years ago by Faris & Dunham (1939) and has been replicated more recently in Bristol and Nottingham (Ineichen *et al*, 1984; Giggs & Cooper, 1987). The Royal College study (Hirsch, 1988) found a correlation of 0.76 between district psychiatric admission rates and Jarman-8 indices in North West Thames. Indeed, a number of other combined deprivation scores such as ACORN, Unit 9, and the Department of the Environment Social Index (but not Standard Mortality Rate (SMR)) correlate equally highly with admission rates (Thornicroft, 1989). The strength of these associations support Hirsch's proposals that the service norms used in planning psychiatric services should be weighted to take explicit account of the extent of local social and economic deprivation.

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Platelet MAO and 5-HT uptake in agoraphobics

Sir: The hypothesised association between neurotransmitter abnormalities and the anxiety-states panic disorder and agoraphobia with and without panic attacks has been the focus of renewed attention since the introduction of the concept with DSM–III (American Psychiatric Association, 1978). The recent report by Flaskos *et al* (*Journal*, November 1989, **155**, 680–685) describes studies of platelet monoamine oxidase activity (MAO) and serotonin (5-HT) uptake in patients with agoraphobia and neurotic depression compared with a control group. Both measurements are relevant to the neurotransmitter hypotheses of these disorders. The findings of elevated MAO activity in agoraphobia are supported by some studies in the literature but not all. While several studies have demonstrated elevated MAO activity in agoraphobia with panic attacks (DSM-III criteria) there are methodological shortcomings with these studies (Norman *et al*, 1988). Furthermore, in the largest study undertaken to date (Khan *et al*, 1986) no difference was found between panic disorder, agoraphobia or generalised anxiety disorder (GAD) and a control group. More recently, Norman *et al* (1988) found no differences in either K_m (affinity constant) or V_{max} (maximum uptake velocity) of platelet MAO between patients with panic disorder or agoraphobia with panic attacks (DSM–III criteria) and normal controls. These conflicting results are difficult to explain but clearly studies in small numbers of patients using diverse methodological refinements correctly require circumspect interpretation.

The finding of increased platelet 5-HT uptake in 'neurotic' depression is of interest in the context of a hypothesised decreased 5-HT availability in depressive illness. Most reports find decreased 5-HT uptake in endogenous depression but no difference from controls in non-endogenous (neurotic (*sic*)) depression (Langer *et al*, 1987) This is again a finding which requires cautious interpretation given the small number of patients involved. In the agoraphobic group it was stated that there was "no association between the 5-HT uptake kinetics and the central symptoms of agoraphobia". It would have been interesting to have known the relationship with panic attacks, particularly as a significant negative correlation between V_{max} of uptake and the tense-panicky dimension of the fear questionnaire was reported ($r = -0\ 44$; $P < 0.05$). We have previously reported an elevated V_{max} of platelet serotonin uptake in panic disorder and agoraphobia with panic attacks (Norman *et al*, 1989). Certainly, these studies have implications for a serotonin-overactivity hypothesis of panic disorders, which is supported by some neuroendocrine challenge tests (Khan *et al*, 1988). It does not necessarily follow that unchanged ^{3}H-imipramine binding in panic disorder supports the unchanged platelet serotonin kinetics. It is notable that dissociation of 5-HT uptake and ^{3}H-imipramine binding sites has been observed. The more likely association between 5-HT uptake and ^{3}H-imipramine binding site is that of an allosteric control exerted by the latter over the 5-HT transporter (see Langer *et al*, 1987). While ^{3}H-imipramine binding may be normal, 5-HT uptake can be altered, a finding we noted in patients with panic attacks (Norman *et al*, 1989).

It is unusual that in the group of agoraphobic patients studied by Dr Flaskos *et al* (1989) the presence or absence of panic attacks is neither commented on nor quantified. This additional information would facilitate the comparison of their results with those that already exist in the literature.

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'Le Suicide'

SIR: I would like to thank Berrios & Mohanna (*Journal*, January 1990, **156**, 1–9) for their cogent and illuminating critique of Durkheim's use of psychiatric texts in *Le Suicide*. The main thrust of their argument is that Durkheim is both 'selective' and 'idiosyncratic' in his choice of psychiatric material and terminology. I am left wondering, however, just how many contemporary articles would stand up to so rigorous an appraisal, as I am sure it cannot be only late 19th century social scientists who are selective and idiosyncratic in their choice of material. For example (and not because of particular disment), many World Health Organisation (WHO) transcultural studies play down or de-emphasise anthropological explanations or accounts of the phenomena they study (Kleinman, 1987).

Drs Berrios & Mohanna have themselves been selective, if not idiosyncratic, in choosing "not to deal, in any way, with Durkheim's sociological views", explaining instead that they will do this in a separate publication. Durkheim's undoubted methodological sleights-of-hand become more understandable when seen in the broader context of his other works. I would suggest that *Le Suicide* can be seen as an, admittedly flawed, sociological and statistical study of suicide rates as an index of pathological forms of the division of labour. This stems from a positivist desire to study rates, rules and types of social phenomena rather than enquiring about (unknowable) individual intentionality. This is one solution to a dilemma not unknown to psychiatrists attempting to make sense of phenomenological data through the use of epidemiology and operational criteria.

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There are none so double-blind . . .

SIR: Oxtoby *et al* (*Journal*, November 1989, **155**, 700–701) raise important points in their significant and elegant article.

Surely journal editors should follow the policy they imply? No article should be accepted for publication, which includes the claim to have been a 'double-blind' drug trial, unless it contains clear data establishing the validity of that claim It is decidedly odd that hitherto, editors have required only the *simulation* of double-blinding, rather than the reality of how the technique is effectively used.

The experience reported by Oxtoby *et al*, of failing to gain publication of their valid and important critiques, raises another most important caution concerning the scientific literature. In my experience there is in practice a significant rate of rejection of articles and letters critical of substantial methodological flaws in studies; a rejection rate that is unrelated to the truth or relevance of the criticisms, or to the quality of the written submission. There may be several reasons for this. Editors and reviewers (consciously and unconsciously) might not be well disposed to submissions that demonstrate serious flaws in articles they have accepted for publication; especially if the authors of the faulty articles are significant establishment figures.

Another problem arises from the attempts to seek 'peer' review. Items obviously need to be reviewed by experts. However, especially within some fields, a regular ring of reviewers develops, whose personal and emotional investment in particular views lead them to urge rejection of contrary ones. One should be very cautious of using reviewers too prominent or dominant within their field.

This was well demonstrated recently, when this *Journal* (Simpson, *Journal*, October 1989, **155**, 565) published a letter in which I criticised some of the many flaws in the burgeoning fad literature on multiple personality disorder (MPD). I have since received numerous sustaining and supportive letters from American readers, delighted to have seen criticism of this sacred cow of the psychosocial literature actually appear in print. In North America, it is almost impossible for such critical views on MPD to appear in print because all such submissions are reflexly sent for review to a small circle of devotees of MPD, who reject them.

As scientists, authors, reviewers and editors, we should value sincere and informed critics far more highly, for they are a valuable but endangered species. Our disciplines need to treasure iconoclasts and there are more than enough of these about.

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Malaria presenting as atypical depression

SIR: We report here a case of a patient with cerebral malaria who presented in the UK with a hysterical stupor occurring in the context of an atypical depression.

Case Report. A 30-year-old woman was admitted under Section 2 of the Mental Health Act (1983) following a domiciliary visit. She had been stuporose for 48 hours and had stopped eating and drinking. On returning from a day trip to the seaside she had been quite unable to get out of the car because of her semistuporose condition and resisted any attempts to move her, even by force Prior to this episode she had complained of bouts of extreme tiredness, depression and irritability for about four months She also had sleep reversal, with daytime sleepiness and nocturnal insomnia. There was no weight loss, but appetite was impaired. Her family history was negative for all psychiatric disorders, but seven years previously the patient had taken an overdose following the break-up of a relationship. Early life and schooling were unremarkable and she had been successful in her career as an information officer At the time of the onset of depression, she had been in dispute with a girlfriend with whom she had shared a flat. In addition, six weeks prior to admission she had got married and reported some difficulties in forming a relationship with her stepson It was thought that her recent life events were sufficient cause for her depression Her general practitioner (GP) had started treatment with imipramine with a good initial response However, she stopped the drug because of side effects and this appeared to coincide with the onset of deterioration in her clinical state and the onset of stupor.

During the admission to hospital, she took to her bed for two days every 3–4 days, complaining of severe exhaustion and sleepiness. She would then have a few days when she appeared to be quite well and took part in ward activities. Even though all routine haematological and clinical tests were normal, an organic cause for her depression was suspected. An electroencephalograph (EEG) recording showed some temporal theta-wave activity, associated with drowsiness and slow-wave activity of 2–3 cycles per second on overbreathing. This was unusual for her age.

The diagnosis of malaria was only reached during the fourth psychotherapy session, when she began to describe her brother's marital problems. Her brother had married a woman in Bangkok and when this relationship had foundered, her brother became depressed. Together with her parents, she visited him in Bangkok to console him. While describing this visit, she added that they had visited a malaria-infested island and, as an aside, commented that she had not completed her course of antimalarials on her return to the UK. On this information a blood film for parasites was done and the film showed trophozoites in the smear. She was then treated with chloroquine for five days and primaquine for two weeks, and felt considerably improved but her depression remained and this only resolved following a course of fluoxetine (20 mg *nocte*).

According to Daroff *et al* (1967), cerebral malaria can present with five different neuro-psychiatric syndromes. In order of frequency, these are: disturbances of consciousness; stupor and coma (most common); acute delirium; acute psychosis or personality change; movement disorder with tremors, and a stroke-like syndrome with focal neurological defects. Redlich & Freedman (1966) highlight cases which are subacute or chronic, where apathy and depression may predominate. Gopinathan *et al* (1982) presented six atypical cases of cerebral malaria where there was neither fever nor parasitaemia. Our patient had episodes of semistupor, yet remained afebrile despite careful monitoring over a four-week period. An organic aetiology for our patient was suggested by the EEG changes, which were similar to the bilateral slow-wave changes demonstrated by Le Mercier *et al* (1969) in their study of 76 cases of cerebral malaria. In our patient the diagnosis proved elusive despite five independent medical assessments, possibly because of a very low index of suspicion and consequent failure to elicit a comprehensive travel history from the patient. Carothers (1953), working in Kenya, estimated that 3% of all first admission cases to mental hospitals were suffering from cerebral malaria. With increasing foreign travel to areas endemic for malaria, psychiatrists in the UK as well as physicians will need to be aware of the possibility of malaria in cases of depression presenting atypically.

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Corrigenda

Journal, November 1989, **155**, 725. A. S. Henderson is the author of *An Introduction to Social Psychiatry*, not the editor.

Journal, February 1990, **156**, 285. S. H. Verma should read S. M. Verma.

A HUNDRED YEARS AGO

Clinical observations on the action of sulphonal in insanity

Dr Mabon records a series of observations on patients suffering from simple melancholia, melancholia agitata, acute and chronic mania and dementia. Sulphonal was administered 119 times on 114 nights, in doses of 15, 30, 45 and 60 grains. Generally 30 grains were required to bring about a quiet and refreshing sleep. It was given at first suspended in mucilage, but later in hot milk and hot gruel, the advantage of the latter menstruum being increased promptness of action. Dr Mabon thinks that the advantages of sulphonal over other hypnotics are the absence of disturbances of digestion, secretion, circulation, and respiration; its easiness of administration, its tastelessness, its odourlessness, and the resulting sleep closely approximating that of nature.

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Journal of Mental Science, January 1890, **36**, 104.

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British Journal of Psychiatry (1990), **156**, 596–597

Reading About . . .

Psychiatry for Undergraduates

What should one offer to undergraduates as recommended reading for the 1990s? Since the question was last addressed in this journal (Mindham, 1982) much in psychiatry has altered. The student needs to be aware of the reorganisation of mental health services, with the closure of many old hospitals and the push into the community. Other changes include a growth in the elderly population, an ever-increasing number of psychotropic drugs, more emphasis on the multidisciplinary approach and the role of substance abuse in mental disorder, and the strengthened relationship between psychiatry and both general practice and other hospital medical services. There has been new mental health legislation since 1982, and the future doctor will also need to adjust to the advent of NHS reforms, with all its ramifications.

Yet despite these numerous developments, some things will probably never change. Undergraduate teaching should still aim to equip students with a reasonable knowledge of psychiatry, but perhaps just as vital, it should allow them to develop interviewing skills and to acquire a 'feel' for the subject. Although talking and listening to patients, and subsequently following their progress, is the best way of achieving these aims, reading a good undergraduate account of psychiatry can be very helpful. Many student textbooks are available, but a limited budget will obviously influence one's spending. I suspect the vast majority of undergraduates will purchase one such volume and use the library for further reading of the subject.

So what should students expect from their chosen text? I believe it needs to be eclectic, thereby offering a balanced account of psychiatry in a comprehensive and up-to-date manner. Which textbooks accomplish this while being stimulating, clear and concise, as well as practical and readable? Among those available, and there are at least a dozen, three are front runners.

Psychiatry in Medical Practice by Goldberg *et al* (1987) aims to familiarise the students of today with the common psychiatric disorders they are likely to see in primary care and general medical settings as the doctors of tomorrow. Its contents are therefore quintessentially 'state of the art' psychiatry, and great emphasis is placed on interview techniques, assessment and management, as well as the interface between physical and psychological illness encountered in much of modern clinical practice. The book is interesting and concisely written, although by no means compendious (virtually nothing is mentioned of community services or psychiatric problems among ethnic minorities, and mental handicap is skimmed over very briefly).

Student Psychiatry Today by Cohen & Hart (1988) is rather more conventional in its approach, but nevertheless clear and comprehensive in dealing with the variety of psychiatric topics. It is purposefully eclectic and readable, while maintaining a style which the student will welcome. The authors (one of whom I shamelessly admit is myself!) include summaries for each of the major chapters as well as an extensive glossary, and the text is liberally sprinkled with case histories, tables and diagrams, all of which should be appreciated by undergraduates. Those chapters on psychiatric history-taking and mental state examination, schizophrenia, affective disorders, liaison and community psychiatry are particularly well dealt with, and there are substantial sections on all of the important subspecialities.

Fish's Outline of Psychiatry by Hamilton (1984) remains the best of the older books. Although rather out-of-date in some areas, it is nevertheless stylishly written and has proved a highly useful short work.

Other standard undergraduate textbooks deserve some mention. *Psychiatry for Students* by Stafford-Clark *et al* (1989) and *Psychological Medicine* by Storey (1986) remain somewhat archaic and turgid for current purposes, despite several revisions and re-editings. *Psychiatry* by Trethowan & Sims (1983) and the *UCH Handbook of Psychiatry* by Tredgold & Wolff (1975) are similarly afflicted. By comparison, Rees' *A New Short Textbook of Psychiatry* (1988) and Rose's *Essential Psychiatry* (1988) are both quite readable, but too elementary for the student's needs. *Lecture Notes on Psychiatry* by Willis (1989) remains an undergraduate favourite and has recently been updated, but is far too brief in content and should be reserved for the back pocket during the week before Finals. Alternatively, for the student who wishes to learn more of the subject than necessary for passing examinations, the best of the postgraduate reference books is probably Gelder *et al*'s *Oxford Textbook of Psychiatry* (1989), although I would not advise actual purchase unless one's budget is particularly robust!

Apart from the standard texts forementioned, what other books may help the undergraduate gain further insight into psychiatry, albeit from a less formal viewpoint? *Psychiatric Differential Diagnosis* by Pfeffer & Waldron (1987) presents a useful and practical way of understanding mental illness at its most basic level, that is, from the patient's symptoms and signs during the interview. Each chapter deals with different complaints (strange ideas, funny movements, hearing voices, etc.), and then discusses the possible underlying disorders which can produce them. Since this is how clinicians think in everyday settings, this compact volume is a worthy contribution to the aspiring doctor's reading list. Maxwell's *An Outline of Psychotherapy for Medical Students and Practitioners* (1986) offers the undergraduate further information about this subject in a concise and interesting manner. It is easy to follow and covers a wide area of theory and practice in a balanced fashion. *Psychiatry in General Practice* by Edwards (1981) is a short text which provides an excellent account of the detection and management of psychological problems encountered in primary care. Although not specifically aimed at medical students, it is nevertheless recommended reading, since at least half of the undergraduate population will eventually enter general practice.

Finally, which books in the world of general literature help to offer some insight into the psychological experiences of one's patients? To understand an empathise with the person who is suffering mental anguish is to perhaps gain that elusive 'feel' for psychiatry. I would recommend four such books, although there are many others the undergraduate may prefer. *I Never Promised You a Rose Garden* by Hannah Green (1964) and *Virtuoso: the Story of John Ogden* by Brenda Ogden & Michael Kerr (1981) both describe psychiatric illness from the sufferer's and family's point of view. This is important for the student (and doctor) to appreciate, as we often tend to see patients only in the context of the cloistered environment of hospital or surgery My third recommended novel is *Portnoy's Complaint* by Philip Roth (1969), an excellent and tragi-comic account of the turbulent mind of an adolescent that has never been bettered. Lastly, there is a new autobiography, *The God Squad* by Paddy Doyle (1989), relating the author's graphic memories of his childhood and early adult life in Ireland: a time filled with chronic abuse and neglect, emotional pain, handicap and extreme deprivation. How Mr Doyle suffered all this and still retained his psychic integrity, individuality and self-respect is simultaneously an indictment of society and a lesson in courage for others.

This selection of books is not intended as a comprehensive reading list, but I hope it will assist the undergraduate in developing a grasp of psychiatry, not only in order to pass Finals, but also to stimulate a further taste for knowledge in this field.

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Books Reconsidered

Clinical Psychiatry: W. Mayer-Gross, E. Slater and M. Roth*

At first sight these three authors may seem an unlikely trio, but in the event their disparate experience and personalities proved to be complementary and the end product of their labours was a coherent and harmonious whole.

Willi Mayer-Gross (1889–1961) had been a professor in Heidelberg, but came to Britain in 1933, one of several eminent continental refugees who enriched the British psychiatric scene before and after World War II. He had been a pupil of Kraepelin, Jaspers, and Gruhle, and was a leading exponent of 'phenomenology', the exact study and precise description of psychic events. Much of the descriptive material in the book (particularly of the psychoses), which was such an outstanding feature, emanated from Mayer-Gross but with his English text edited by Slater.

Eliot Slater (1904–1984) was a commanding figure, both physically and intellectually. His main research interest was psychiatric genetics, although he also made important contributions in neuropsychiatry and the neuroses. He first met Mayer-Gross while training in genetics in Germany, and shared his phenomenological approach and Kraepelinian stance. His style of writing was an extraordinary amalgam of richness, robustness and precision. Slater was responsible for all the genetic sections and played the major role in the writing of the controversial Introduction, with its polemical critique of psychoanalysis.

Martin Roth (b. 1917) trained initially in neurology, but developed an interest in psychiatry partly through the influence of his chief, Russell (later Lord) Brain. He attracted the attention of Slater, who was impressed by his 1948 publication in Brain on hereditary ataxia. Shortly afterwards he went to work with Mayer-Gross at the Crichton Royal Hospital, Dumfries. Mayer-Gross was invited to write a review of old age psychiatry for the 1950 *Recent Advances* series but, finding himself fully occupied in research and lecturing, he asked Roth to take on the task in his stead (from little acorns...). As a result, Roth was invited to write the chapter in *Clinical Psychiatry* on 'Mental disorder in trauma, infection and tumour of the brain' and 'Ageing and the mental diseases of the aged'. In addition, he contributed to the critique of psychoanalysis, and added the recommended "multi-dimensional approach", part of the chapter on 'The epilepsies', and the sections on anorexia nervosa, anxiety, and hypochondriasis in Chapter IV. The whole text was, however, seen and discussed by all three authors. Roth's command of language equalled that of Slater but with a style of distinctive elegance. He was the great drawer together of threads from many sources into a rich tapestry of original thinking, best exemplified in the 1954 edition by the early exposition of his then revolutionary but now commonplace separation on prognostic grounds of the different syndromes of old-age mental disorder.

The Introduction, which firmly wedded psychiatry to medicine and expressed scepticism about the relevance of sociology and anthropology, aroused most controversy and not a little opposition by its critiques of contemporary schools of psychiatry, particularly those of Freud and Meyer. Comments like, "support for the [Freudian] theory is won, not by producing evidence in its favour but by enlarging the circle of those who believed in it", and, "Meyer's enthusiasm in the fight against Kraepelinian classification led him to throw out the baby with the bath water", were not intended to be emollient, nor were they perceived as such. To some they were a clarion call to a more scientific psychiatry, to others an unbalanced overreaction. As one reviewer remarked, the authors could not be accused of fence-sitting.

There were a number of unusual features for a book of the time, including detailed case-taking schemata for ordinary psychiatric, uncooperative or stuporose, and organic patients, chapters on 'Mental deficiency', 'Child psychiatry' and 'Administrative and legal psychiatry' (both at home and abroad), and an extensive bibliography. Chapters on 'Symptomatic psychoses' and 'Chemical intoxications and addictions', including reference to amphetamine, morphine, cannabis, and mescaline, have a prophetic ring.

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*London: Macmillan (1954). Second edition 1960, third edition 1969, reprinted with amendments 1977

The 1954 edition is also of interest as a historical document, providing a glimpse of the practice of psychiatry before the psychopharmacological revolution. Recommended treatments now seem crude and often naive. Thus, paranoid reactions were treated with either superficial psychotherapy or leucotomy, and depression was treated with ECT or awaited spontaneous remission. Agitation or insomnia might respond to Tinct opii. The conventional treatments for schizophrenia of insulin coma, ECT, and leucotomy were described, but the authors were clearly not happy with them, for insulin coma was called "crude amd empirical" and, referring to leucotomy, they remarked "there is no case for a wholesale handing over of chronic schizophrenics to the knife of the surgeon". The 1960 edition brought the first mention of the use of neuroleptic and anti-depressant drugs and lithium, and by the 1969 edition treatments of all kinds had become more numerous and sophisticated.

Successive editions saw also a toning down of the anti-Freudian polemic of the 1954 edition, and by 1969 some value was allowed to Freud's contributions in modern psychiatry. By 1969 the Schneiderian view of neuroses as developments from personality deviations, originally propounded by Mayer-Gross, was modified to a more syndrome-based approach at the hands of Roth. He also revised the section on paranoid psychosis, extended considerably Mayer-Gross' chapter on 'Alcoholism, drug addiction and other intoxications', and added chapters on 'Exogenous reactions and symptomatic schizophrenias' and 'Social psychiatry'.

It is difficult for young psychiatrists of today to appreciate the impact this novel, stylishly written textbook had on those, like myself, who were taking their first faltering steps in psychiatry in the 1950s. For the first time the written text seemed to chime with everyday clinical experience. The authors' views that psychiatry was afflicted by sectarianism and unwarranted attempts to expand its scope and was in danger of becoming isolated from medicine, and their prescription of a return to the bedside and the discipline of clinical observation, struck a chord in many contemporary seekers after psychiatric truth.

Translated into many languages, its impact overseas was no less profound. It brought succour to the beleaguered minority of biological psychiatrists in the USA, and prominent figures such as Robins, Guze, Klerman, and Spitzer have acknowledged their indebtedness. Indeed, a connection can be traced from the book's multi-dimensional approach to the development of modern operational definitions of mental disorder, such as the Research Diagnostic Criteria and DSM–III.

For over two decades the book was, for many, the psychiatric bible, and even today it has not lost its value as an authoritative reference work, for it is still one of the most frequently cited textbooks in scientific papers. The high esteem in which it was held is conveyed by E. W. Anderson's review of the 1969 edition: "So much praise has been justly accorded this book that there is little the reviewer can add. This is the most outstanding textbook of psychiatry in English, certainly the reviewer knows of no other English work of the kind to rank with it. The weight of scholarship behind it is in itself impressive It may be said fairly that the book is an outstanding contribution to British psychiatry".

Dare we hope for a fourth edition? Rumour has it...

Acknowledgement

I am indebted to Professor Sir Martin Roth for providing much background information on the compilation of the textbook

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Book Reviews

Editor: Sidney Crown

Contemporary Themes in Psychiatry: A Tribute to Sir Martin Roth. Edited by KENNETH DAVISON and ALAN KERR. London: Gaskell. 1989. 532 pp. £20.00.

I suppose the idea of a book of essays or articles brought together in honour of some great scholar on his 70th or 80th birthday is a specifically German one since we call it a *Festschrift*. Sir Martin Roth is 72, and undoubtedly the greatest living British psychiatrist: heir to Kraepelin, creator of the Newcastle school and founder of the Department of Psychiatry at Cambridge. Here is a book of over 500 pages and nearly 50 articles contributed by former pupils, colleagues and friends to mark his 70th birthday.

Such a book is tricky to produce because of its multiple and ambiguous functions. Is it a private birthday card expressing love and esteem, or a public salute trumpeting the hero's contributions to national and international culture? Should the editors invite everyone to write something, for fear of offending those left out, or limit it to pupils only, or eminent colleagues only, or those with something new to say? Must the topic of each paper touch the hero's own work and interests at some point, or will anything go? (Too often contributors to a *Festschrift* dig out some old bit of unpublished research, or a paper previously rejected by a journal, or else offer a paper that only a specialist would want to read as their signature to the birthday greetings.) Moreover, loyalties apart, will anyone actually buy the book?

Davison and Kerr have done a good job. First, there is a core of general essays everyone can read with profit, or will be glad to have to hand. Examples are: Brandon (an old pupil) on "anxiety: does classification help?"; Shaw (a neurologist colleague) on "Psychiatric morbidity following cardiac surgery"; Gelder (Oxford) on "Cognitive pathology and cognitive treatment of depressive and anxiety disorder"; Ploog (Munich) on "Psychopathology of emotions in view of neuroethology"; and Kiloh (an old pupil) on "Psychosurgery and deviance". Then, there are groups of papers around three of Sir Martin's major contributions to clinical research and practice: the differential diagnosis of depressive illnesses, the psychiatric disorders of old age, and questions of abnormal movements (tardive dyskinesia, akathisia).

With his collaborators he set out to subdivide depressive illness into groups distinguished by different clusterings of depressive symptoms, different outcomes, different therapeutic responses and probably different aetiopathologies. This proved difficult, controversial, and disappointing. However Carney (an old pupil) offers a review of further evidence to date supporting the original division into endogenous and neurotic depression, while Akiskal (USA) describes new studies on atypical or anxious depressions and dysthymic states. Cassano (Italy) shows that the introduction of symptom sequencing (bipolar I, II, etc.) improves delineation of subtypes, and Andreasen (USA) rightly emphasises that the scientific future lies in subdivisions of depression, even if the way there is not yet clear.

Thirty years ago, Sir Martin was a pioneer in the study of mental disorder in old age. His recognition of late paraphrenia, a treatable condition as it turned out, is updated by Kay (an old pupil); the importance of deafness in the aetiology of mental illness is reviewed by Cooper (an old pupil); while Day (an old pupil) describes the Newcastle studies on the aged mentally handicapped. Roth's more recent work on dementia also gets discussed in several papers. There is no space to refer to other epidemiological or biochemical work or the stimulus to child psychiatry at Newcastle.

I have said enough to show that this book needs to be widely available in libraries, and is worthy of personal purchase. It is very much more than just a tribute to a leader. Wide as its scope is, however, I wish it had given a fuller biography of Sir Martin, or possibly some additional papers on other aspects of his life's work. That he has been a philosopher, editor, and co-author of the most important textbook of psychiatry in English in the past 50 years is perhaps by the way. However, that he has been an influential adviser of Government, and above all, a creator of our Royal College, with all its work in improving training and standards of practice, is something that would have been valuable to note.

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Freud. By ANTHONY STORR. Oxford: Oxford University Press. 1989. 135 pp. £3.95 (pb), £14.95 (hb).

This is an admirable little book: the author succeeds in giving a concise and lucidly written overview of Freud's work and life in no more than 128 pages. Furthermore, this is no mere summary: the approach is constructively critical without minimising Freud's great achievement. As we would expect from Storr, who has been concerned with the psychoanalytical aspects of creativity for a long time, he is particularly good at questioning Freud's theory of art. There is a skilful and revealing summary of Freud's case histories which shows how much more important the exploration and understanding of the human psyche was for Freud than its therapy.

There are two critical comments I wish to make. Firstly, Storr refers to "Freud's frequent use of the word 'instinct'". In fact, Freud very rarely talks about 'instinct' and when he does he uses it in the classical sense of inherited and stereotyped behaviour, mainly ascribed to animals. Freud refers to 'drives' (*Triebe*) which are infinitely mouldable: there could be no sublimation of instincts. It is a pity that Storr has allowed himself to be misled by Strachey's mistranslation. It is time that writers about Freud rid themselves of the distortions Freud has suffered from the hands of his English translator My second comment touches on the question: for whom is this book meant? It seems to have been written for a reader who knows very little about Freud and the fact that it has no bibliography (which I regret) should probably be seen in the light of this. On the other hand, the numerous quotations of Freud refer the reader to the Standard Edition which is hardly within easy reach. I hope this inconsistency is looked into when the book is reprinted as it certainly deserves to be.

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Neuronal Receptors, Endogenous Ligands and Biotechnical Approaches. Edited by M. E. VARTANIAN. Madison: International Universities Press. 1989. 149 pp. $35.00

The aims of this book are laudable, but the volume gets hoisted by its own petard. In the preface the editor states that "there is a constant need for a neurobiologist to be informed about current research and advances in adjacent branches of brain science" and that "the high momentum in this field [leads to] a strong need to publish monographs that would review the results of up-to-the-minute most promising brain studies". Quite so, but the rub is that this volume has no reference more recent than 1986. Lag-times in the publication of books means that to obtain reviews of significant developments, the interested neurobiologist is better advised to turn to journals such as *Trends in Neurosciences* and *Trends in Pharmacological Sciences.*

The reviews in this book with Russian, (other) European and American authors (in a ratio of 4/3/1) are variable in terms of breadth, depth and quality. There are three chapters on the neurochemistry of long-term antidepressant administration and five on the gamma-aminobutyric acid (GABA)/benzodiazepine receptor and in each the message is different and the consequent picture confused. The introductions and discussions are the best parts of the book but are often too short to be useful.

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Psychiatric Dictionary (6th edn.). By ROBERT JEAN CAMPBELL. USA: Oxford University Press. 1989. 835 pp. £35.00.

So Greta Garbo had eremophilia and Manuel Noriega is a cratomaniac. That's the trouble with dictionaries. They're at their most intriguing when they're at their most trivial.

It is hard to say why lists of words and their meanings make entertaining reading but they do, and Campbell's list of psychiatric words is no exception. Perhaps the pleasure of dictionaries is the pleasure of soap opera. there's no real plot, the format never changes and you can dip in anywhere without feeling disadvantaged by what you've missed out.

In preparing a psychiatric lexicon it must be hard to decide what to miss out altogether once the key terms in current use have been defined. What should be done with all those philias, phobias, manias and syndromes which are too obscure to be useful? Campbell includes them all, and fascinating they are too. Have you heard of biting mania, a compulsion to bite fellow nuns which spread through the convents of Northern Europe in the 15th century? Did you know that someone whose life centres round their computer may suffer from machine fever? And, for all I know, one of the symptoms may be chattering mania, defined here as an "uncontrollable urge to talk gibberish".

Over the 800 or so pages, clear summaries are presented of important concepts, both old and new. But the recent language of molecular genetics is only partly included and, while modern practices such as cognitive therapy are briefly treated, old hat like Rorschach donates a disproportionate number of items. Beside these lies an epic list of curiosities, from which I discovered Skoptsy, a Russian sect which believes in self-castration, and the Grantham lobotomy, previously unknown outside the Cabinet.

Occasionally the concise style sounds like anti-academic parody. There is, for example, the enjoyable cross-reference: marital infidelity – see paranoia, conjugal. Then comes a remarkable definition of the sexual practice 69, in which "the head of each person is near the feet of the other". The *feet*? Well, now you know.

But it would be wrong to suggest criticism of Campbell, who has produced a fine volume for both checking and browsing The pressures of precise definition are already enough for most lexicographers, as illustrated by one famous story, itself concerning paranoia, conjugal.

The story goes that Noah Webster who in the 19th century produced the massive American dictionary that still carries his name, was, while engaged in this project, discovered by his wife, *in flagrante* with the chambermaid. "I am surprised", cried the wife. "No", replied Webster, "*We* are surprised. *You* are astonished."

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The Myth of Atlas: Families and the Therapeutic Story. By MAURIZIO ANDOLFI, CLAUDIO ANGELO and MARCELLA DE NICHILO New York· Brunner/Mazel. 1989. 238 pp.

If I had not had to review this book, I would not have got very far. Yet I'm very glad I finished it. Andolfi, a professor of psychiatry in Rome, is a distinguished family therapist who has had a reputation for wild innovation, almost as if there were no other way to get attention while the brilliant work of the Milan school was going on nearby This book, whose untidiness reveals its triple authorship, represents a more mature approach than I had expected. Reading it has given me renewed courage in the search for an independent discipline of family therapy. These authors draw happily and openly from all kinds of therapy styles and schools, yet seem to have a way of their own. They are true eclectics. Most stimulating are the verbatim passages of therapy sessions, which often read like something by George Bernard Shaw.

There are two problems with this book. Firstly, it was originally written in another language, and this shows in, for example, the fact that a lot of the plays on words have to be explained (e.g. the subtle double meanings in *tempo* and *storia*). Secondly, the authors are trying to make a theoretical statement using language that is simply not interesting, and is often very difficult to follow. This is a common problem in family therapy writing, and is what put me off at the beginning.

From the therapy examples, however, it is clear that it would be very enjoyable, interesting and instructive to spend time with these colleagues at work. They combine a sense of respect and affection for suffering individuals with inspired and effective playfulness A central clinical task is to find a powerful metaphor – a throne, a doormat, Atlas – which is then used again and again. They follow up their cases and often find that these metaphors are remembered for years afterwards. There are many thoughtful comments which I will remember, too: "that therapy begins at the moment it ends"; and "that the therapist is the director of the therapeutic story", an important reminder of that particular kind of activity, related to work in the theatre, which family therapy always demands. A new character joins the action towards the end, that great American performer, Carl Whitaker, well known for his use of the absurd. He acts as consultant to the therapists and the 'Atlas' family whose story is narrated in the final chapter.

This is not a book for beginners They do not really tell you how to do it, nor indeed how they do it. Although refreshingly aware of the vulnerabilities of the therapist, the authors do not show much of their actual thinking, as opposed to theorising, that leads to the interventions they make – another reason why meeting them might reveal more of their secrets than a book can do

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Obsessive-Compulsive Disorder in Children and Adolescents. Edited by JUDITH L RAPOPORT. Washington, DC· American Psychiatric Press, and Cambridge: Cambridge University Press. 1989. 355 pp. £35.00.

Obsessive-compulsive disorder (OCD) is a fascinating syndrome that is attracting increasing psychiatric attention. It may hold the key to our understanding of many thinking processes. Sufferers and their families can be crippled as much as in chronic schizophrenia, yet they can now be helped to overcome much of their disability within a few months.

Most studies of OCD have been in adults, so systematic work in children is to be welcomed. The present volume is a collection of papers from the USA, many from the Child Psychiatry Branch of the National Institute of Mental Health (NIMH), which is headed by Rapoport. The topics cover a wide range· phenomenology; diagnosis; cognitive, behavioural, neuropsychological, psycholinguistic and neurological assessment; drug and behavioural treatment; epidemiology; ethology; superstition; religious scruples; and basal ganglia dysfunction.

An original chapter in the book is that on ethology by Swedo. She suggests that OCD involves the expression of complex, fixed-action patterns similar to those noted by ethologists, but with inappropriate release and lack of inhibition. We understand this lacuna little better than William James did a century ago, who noted that in OCD the 'fiat' marking the end of certain sequences is lacking. However, we do have pointers to some relevant mechanisms being located in caudate, limbic and associated structures. The evidence on brain mechanisms is reviewed by Swedo in a later chapter.

Childhood rituals and superstitions, and their differences from those of OCD, are well discussed by Leonard. Religious scruples in relation to OCD are nicely reviewed by Suess & Halpern. A careful epidemiological survey of adolescents by Flament *et al* yields a minimum point prevalence of OCD of 0.35%. The finding of a male preponderance in early-onset cases recurs in the papers of others from NIMH.

The treatment section is a bit outdated and uncritical. It acknowledges the importance of behaviour therapy for OCD but not the redundancy of therapist-accompanied exposure, nor the centrality of self-exposure homework which includes recording in diaries. A family group is described with neither outcome data nor guidance on how to help relatives become exposure co-therapists.

Serotonergic drugs, especially clomipramine, are lauded with scant regard for the paucity of evidence about other antidepressants. Case histories in a family suggesting the value of imipramine are dismissed. Medication is called 'effective' despite much relapse on stopping it, potential problems of long-term medication in young people, and the NIMH finding that initial responsivity to clomipramine fails to predict outcome two to seven years later.

The book cites diverse old sources but is parochial on recent work. The ethology chapter could have linked its ideas with relevant work on preparedness and on learning models of rituals carried out in Scandinavia and the USA (Öhman, Lang, Bolles & Mineka). Neurophysiology discussions omit Gray's idea about failure of a septo-hippocampal comparator system (UK). The chapter on superstitions leaves out tantalising work by Rabavilas (Greece). That on religious rituals misses important ideas from Greenberg (Israel). A review of behavioural assessment omits the often used Compulsion Checklist (UK). Discussion of behavioural treatment omits most of the many studies in the Netherlands, the UK and Greece.

More careful scholarship and editing would have improved the book. It is padded by 50 rambling pages of patients' autobiographies. An article published in 1986 is not really 'in press'. Authors' names are misspelt and initials are wrong. There is no author index. The subject index is skimpy. Nevertheless, some of the chapters are valuable and make the book suitable for libraries

ISAAC MARKS, *Professor of Experimental Psychopathology, Bethlem-Maudsley Hospital and Institute of Psychiatry, University of London*

How to Do Self-Analysis and other Self-Psychotherapies. By LOUIS A. GOTTSCHALK. London: Jason Aronson. 1989. 202 pp. $25.00.

The fact that this review is published by the *British Journal of Psychiatry* rather than the *Readers' Digest* perhaps reflects the far-reaching impact of Thatcherism. This brief consumer guide is written specifically for lay readers in an attempt to take the mystery out of the process of psychotherapy and to introduce a variety of self-help procedures designed to improve the quality of life and to help the individual cope with the problems of human existence.

These procedures include self-analysis (covered in 69 pages), meditation, conditioning and behavioural methods, religious approaches, autogenic training, self-hypnosis and finally, self-psychotherapy with crystals (covered in four pages).

Over half of the section on self-analysis deals with dream analysis but basic psychoanalytic principles are well described, reflecting the author's analytic training, and Freud's own reservation about self-analysis is included. The behavioural approach seems better suited to the format of self-help and clinicians who give their patients audiotapes and pamphlets on relaxation techniques may find additional useful material here

The section on religious approaches includes a review of a study of religiously mediated change in homosexuality and ends with an account of a patient with metastatic cancer who used faith-healing ceremonies Teachers of crystal therapy advise you to let you crystal rest among potted plants. A state of not discriminating between something alive and not alive is fostered by you being told to introduce your crystal to your favourite places and to your favourite seasons of the year. This awakens the magical intuitive child in you and promotes what Freud called primary process thinking. The author states, however, that the healing effects of crystals are illusory but that the symptomatic relief available through such methods should not be overlooked The author also states that there is no risk involved in trying out these methods on oneself but wisely concludes with an appeal for systematic studies.

As psychiatry moves towards a consumer-orientated approach under the White Paper, perhaps it is important that we are all aware of guidelines being offered to the general public – individual guidelines which are likely to increase and which require careful monitoring.

JIM WATTERS, *Senior Registrar in Child and Adolescent Psychiatry, Tavistock Clinic, London*

The Technique of Psychotherapy (4th edn), Volume 1 & 2. By LEWIS R WOLBERG. London: Harcourt Brace Jovanovich. 1989 768 pp & 869 pp. £87.25

Lewis Wolberg died in 1988 but not before he was able to put the finishing touches to this, the fourth edition of his major psychotherapy text. Wolberg and his wife founded a community treatment centre in New York in 1945. This was a successful, pioneering venture which attracted therapists with diverse orientations and engendered a series of training programmes. The first edition drew together what was known about the ingredients in effective psychotherapy: this emphasis has been the informing principle in subsequent editions His work, however, is not in the integrative mode of Jerome Frank

Rather, he surveys in encyclopaedic style the major modes and schools of psychotherapy in their current form, presents their key theories and practices, and, most usefully, makes constructive criticisms.

The subject index spans topics from creating motivation for abstinence from alcohol through adoption, behavioural prostheses, frigidity, logotherapy, recording sessions and terminal illness to zoophilia. Volume 1 offers several definitions of psychotherapy, has three long sections on supportive, re-educative and reconstructive therapy, and then includes a detailed consideration of the beginning and middle phase of therapy, the latter spilling over into the second volume Volume 2 details the issues to be considered in the terminal phase of therapy, adjunctive aids including physical treatments and informative books that may be recommended to patients, short-term psychotherapy, emergencies, supervision, the use of computers and untoward reactions (i.e. negative effects).

In my review of the third edition, I compared psychotherapy to music: the score can be written but the realisation demands creative interpretation. I looked to see how such an experienced practitioner would, in the endeavour to be comprehensive, also convey his essential human concern as a therapist. The results of my search were somewhat disappointing. On this occasion, however, there is a clearer sense of what was important for Wolberg. A psychodynamic way of thinking about what goes on in psychotherapy is central to his approach, as is the drive in a non-doctrinaire way to harness what successful therapists do in helping patients to achieve both symptomatic relief and sufficient self-understanding in order to bring about some personality change.

Successful therapists rapidly establish rapport. They develop a theory about the patient's problems and from this devise a treatment plan in collaboration with the patient. Unrealistic expectations are challenged, steps are taken to enhance motivation and the plan is tailored to individual needs. They attempt to work within the framework of the patient's belief system but are able to confront distorted cognitions, evasions and resistances, the self-defeating residues of past experience. Countertransference feelings are recognised and controlled; the more intuitive therapists use them to further their understanding of the patient's mind. Constructive behaviour is reinforced. When, as is generally the case, the aim of therapy is not personality reconstruction, the therapist brings the therapy to a close at a point when the patient can manage on his own, thus avoiding the therapeutic stalement of dependency. Apart from seeing psychodynamic theory as being the most profound vehicle for understanding psychological problems, Wolberg avoids the trap of having a cherished theory. Instead, he delineates the strengths and limitations of different theories and approaches and encourages the able therapist in the selective use of what is known in the service of his patient

Changes in emphasis since the third edition appeared 11 years ago document important alterations in psychotherapy practice: the move towards brief therapy, often with a symptomatic focus, the search for clarity in identifying important trends or patterns in personality and in the therapeutic focus; the growing prominence of cognitive behaviour therapy with its stress on contracts, altering cognitions and homework assignments; and, finally, a greater valuing – although still only at a superficial level – of research findings and a recognition of the need to find appropriate methodologies.

These two volumes can be confidently recommended to the psychotherapy teacher and to the keen student who wants to enhance their knowledge in breadth as well as in depth. The wisdom that they contain should also find a place on library shelves.

MARK AVELINE, *Consultant Psychotherapist, Nottingham Unit*

Child Psychiatry and the Law. Edited by DORA BLACK, STEPHEN WOLKIND and JEAN HARRIS HENDRIKS. London. Gaskell. 1989. 178 pp. £10.00.

This excellent book reviews child psychiatry practice in relation to legal proceedings involving children in England and Wales. It is a practical book, outlining in each chapter the issues involved in particular areas of reporting. Each chapter ends with a checklist for those preparing reports. The areas covered are extensive, ranging from the actual preparation of reports to issues of confidentiality, custody and access, child abuse and neglect, placement and compensation claims. The concluding section of nearly 60 pages consists of actual reports. These are reproduced, warts and all, as examples of real forensic practice, balancing the theoretical issues dealt with in earlier chapters. There is a brief glossary of legal terms which might perhaps have been excluded or extended.

Overall, the book is a good review of practice in this field and an excellent discussion of the areas of required involvement of child psychiatrists in legal practice. It is obvious from reading the chapters that the authors have extensive forensic experience, but the honesty of the specimen reports brings them down to earth for the day-to-day practising child psychiatrist. Child psychiatry and the law fit uneasily together, but since both have as their prime aim the 'best interests of the child', then the constant and recurring theme of the 'least detrimental alternative' for children is one which both lawyers and child psychiatrists might well share. I felt that the best chapters were those on placement issues and child abuse and neglect.

My main criticisms are that there is no index and that, although there are references to Scottish law, there is no separate chapter which refers to the differences in the Scottish legal system. Such a chapter might have clarified some of the issues raised, particularly in terms of juvenile justice. These are, however, minor quibbles and I think that this will become a standard book for teaching about the legal aspects of childhood. The authors are to be congratulated in being clear, concise and honest in their approach to contentious matters. I recommend this book for trainees in child psychiatry and their senior colleagues, and to social workers and others working in fields adjacent to child psychiatry. Perhaps even adult psychiatrists might understand the position of child psychiatrists better if they read this book, as might lawyers before they ask for reports on children.

LES SCARTH, *Consultant Child Psychiatrist, Department of Child and Family Psychiatry, Royal Hospital for Sick Children, Edinburgh*

Hartland's Medical and Dental Hypnosis (3rd edn). Revised and updated by DAVID WAXMAN. London: Baillière Tindall. 1989. xvi+501 pp. £19.95.

John Hartland wrote as he spoke, using the first and second person singular to invite the reader to a personal tutorial alongside the psychiatric couch. Although as a second author, Waxman could obviously not continue in this style, he has managed to maintain Hartland's clarity of description as well as the original author's remarkable ability to communicate his ideas and therapeutic methods.

We have in this third edition virtually a new book comprised of rearranged hard-core Hartland along with a précis of some recent advances in hypnotherapy and allied subjects. The result is a well set-out volume which wisely retains and up-dates all the practical information of the original editions and thus ensures a continuance of its success as a teaching handbook.

Additional practical material introduced by Waxman includes his 'ego-assertive retraining' technique, which, as he acknowledges in the book, is a refinement of both Hartland's 'ego-strengthening' and the 'assertive training' of Wolpe and Lazarus. This technique also utilises Wolpe's 'reciprocal inhibition' method, used in the treatment of anxiety states.

In compliance with his new chapter "Some dangers of hypnosis", Waxman has sensibly reverted to a 'no touch technique' in the induction and deepening of the hypnotic state. For example, in the second edition a method of deepening by the induction of limb heaviness is described in which the patient's arm is gently stroked from shoulder to wrist. In the third edition the limb heaviness is induced by verbal suggestion alone, which eliminates any possibility of the stroking manouevre being misinterpreted by the hypnotised patient as an assault or sexual advance.

As in the previous editions, theory has been kept to a minimum and where any additional theoretical material has been presented, its explanation has remained at a superficial level. In the majority of cases, sufficient references have been included to enable the readers to extend their knowledge. However, the most important development in the understanding of hypnosis has been the neurophysiological research into altered states of consciousness and asymmetrical brain function The covering of this subject in the chapter "The nature and theories of hypnosis" could be improved and the references are inadequate. I would have liked to have seen included some discussion on the hyperventilation syndrome and its relation to hypnosis, and similarly some mention of the treatment by hypnosis of the Multiple Personality syndrome as defined in DSM-III-R.

These are all minor criticisms: the third edition of Hartland's book maintains and improves its *raison d'être*. It continues to be one of the most comprehensive books of practical instruction in the use of hypnosis in medicine and dentistry available today.

DAVID L. PEDERSEN, *Lecturer, British Society of Medical and Dental Hypnosis, London*

Textbook of Pain. Edited by P. D. WALL and R. MELZACK. London: Churchill Livingstone. 1989 1080 pp. £95.00.

The second edition of this most comprehensive textbook on pain follows the first edition by five years. There are many new contributors and a few chapters including those on back pain and the failed back and a chapter on pain in children. The new chapter "Movement education and limitation of movement" looks at the physiotherapist's role in rehabilitation and improvement of function, often crucial in the management of those suffering chronic pain.

The introduction by Professor Wall examines the definitions of pain, and the effects of injury on the peripheral and central nervous systems, exploring them not as hard-wired circuits but as systems that change progressively with time. The developing approach to pain management is expressed in the final sentence that "Our task is not so much to cure pain as to aid recovery".

The book is divided into three sections. Section one looks at the basic aspects of physiology and pharmacology of pain mechanisms and pathways. Psychological aspects are also examined in terms of emotion and cognition. This section ends with three chapters on the assessment of pain in both experimental and clinical situations. It is difficult to cover all aspects of the assessment of pain patients in a single chapter, and the emphasis is on the objectives and limitations of some methods. Section two deals with clinical problems in which pain predominates. These chapters give thorough accounts of the aetiology, diagnosis and treatment of pain related to a particular area or organ system As well as the familiar chronic pain problems, this section also looks at a number of acute pain problems including pain following trauma and the pain of burns. As one would expect, the chapter by Bonica & Chadwick on labour pain is excellent, although it is unfair to single this out. A subsection is devoted to carcinoma and includes chapters on psychological and social aspects of cancer pain, and pain and impending death. Section three examines therapeutic aspects and covers the whole spectrum ranging from heat treatment to brain stimulation A whole chapter is now devoted to acupuncture. A subsection looks at psychotherapy and includes a wide range of treatment modalities. Throughout the book there is an emphasis on a multidisciplinary approach to pain management.

The illustrations and tables are generally clear and the photographs of high quality. All chapters are well referenced and the index is comprehensive. It is difficult to find fault. This book will be well known to specialists with an interest in the management of patients with chronic pain. It can be strongly recommended to most other specialists, who will undoubtedly find many chapters worthwhile reading, and the book should be available to all practitioners for reference

DAVID G. GAYLARD, *Consultant, Pain Relief Clinic, Royal Devon and Exeter Hospital, Exeter*

Depression: An Integrative Approach. Edited by KATIA HERBST and EUGENE PAYKEL. Oxford: Heinemann Medical Books. 1989. 249 pp. £25.00.

This book can be reviewed in one word – superb! It covers several major aspects of depression, using experts in each area. The approach is stimulating, erudite and easy to read, a combination seldom found in the more usual writings of experts. The four major aspects dealt with include the extent and nature of the disorder, the causes of depression at specific times of life, and the management of depression Within each of these broad areas there are chapters on specific topics. These include the social causes of depression, genetic and neuroendocrine aspects, drug-related depression, depression in the puerperium, and cognitive treatment for depression, to mention but a few. One of the strengths of this book is that the referencing is up to date and includes references to papers published last year. Especially praiseworthy is the clarity of the writing, and chapters such as drug-related depression, suicide and the management of suicide attempts, and depression in the elderly I found particularly easy to read. An unusual and welcome chapter is that dealing with self-help groups in the management of depression This is an area which is under-researched and of which psychiatrists have little more than impressionistic knowledge. I learnt a great deal from that particular contribution. In addition some useful addresses were provided at the back which will no doubt be of assistance also.

The only slight criticism I have is the failure to mention adjustment reactions. This is particularly disappointing since there is a detailed chapter on the recognition and diagnosis of depressive disorder by general practitioners. While I realise that this book is dealing with depressive illness, it is an unfortunate oversight not to mention this group of conditions, lying as they do in the hinterland between distress and disease.

This book is an essential in every library and should be on every psychiatrist's bookshelf.

PATRICIA CASEY, *Consultant Psychiatrist, Professorial Psychiatric Unit, Cork Regional Hospital*

The Instruments of Psychiatric Research. Edited by CHRIS THOMPSON. Chichester: John Wiley. 1989. 345 pp. £35.00.

This is an excellent book which will be of great assistance to those planning research projects. To my knowledge this is the first time that reviews of so many symptom and diagnostic instruments have been gathered together in one volume

Chapter 1 is a brief and clear description of the scientific aspects of measuring symptoms and clinical states, together with a commentary on validity, specificity and sensitivity. Chapter 2 is an overview of the RDC, DIS, DSM-III and PSE systems; it is up to date and gives a relevant history of the development of these important systems. Chapters 3 to 13 cover symptoms in schizophrenia, affective disorders, anxiety, eating disorders, suicide, drug and alcohol abuse, personality disorder, social, child and old age psychiatry. All of these chapters are well written and succinct. The chapter on the classification of personality disorder is particularly welcome and is a valuable contribution to a difficult area of research.

I recommend this book for medical trainees in psychiatry as well as for any research workers.

HUGH GURLING, *Senior Lecturer, Molecular Psychiatry Laboratory, Department of Psychiatry, University College and Middlesex School of Medicine, London*

Handbook of Counselling in Britain. Edited by WINDY DRYDEN, DAVID CHARLES-EDWARDS and RAY WOOLFE London: Routledge. 1989. 470 pp. £30.00 (hb), £15.00 (pb).

This edited book consists of 25 chapters written by 27 contributors and grouped together into five parts. The introductory section contains two chapters, the first of which provides some useful information about the organisation of the British Association of Counselling, which was involved with the publication of the book but the nature of which is never made clear. The second part discusses counselling as practised with individuals, couples, families and groups. In view of the more abstract level at which this material is dealt with, it would have been more assimilable if it had been presented later. The next section outlines the way in which counselling is conducted in the main sectors of social work, primary care, education, and work. This is followed by the largest portion of the book which considers a wide variety of concerns that may arise in counselling such as race, gender, religious faith, careers guidance, bereavement, AIDS, and drug problems, among others. The book concludes with a chapter on research (or rather the paucity of published work in Britain) and on the issue of professionalisation. The lamentable scarcity of well conceived research may persist until counsellors themselves are actively encouraged to pursue it, or at least be willing to participate in it.

Unfortunately, in a text of this magnitude and nature, very little attempt is made to outline the main aims of the book, to justify or even to explain the organisational basis for the wide range of material covered, or to try to highlight and draw together some of the major similarities and differences in the diverse contributions The stipulation to authors that the content of their chapters should be ordered in terms of the three rubrics of principles, issues, and future developments does not guarantee, and did not in this case, the coherence of the material. Somewhat surprising in view of the size and role of organisations such as Relate and the Samaritans is the small amount of discussion of relationship difficulties and suicidal behaviour. Nonetheless, despite the restricted space allotted to them, many contributors have provided a useful introduction to the problems they confront and the context in which they work.

DUNCAN CRAMER, *Lecturer in Social Psychology, Department of Social Sciences, Loughborough University of Technology*

Handbook of Behavioural Family Therapy. Edited by IAN R. W. FALLOON. London: Unwin Hyman. 1988. 485 pp. £30.00.

This is one of those rare reference books which will appeal to both the specialist therapist and general professional alike. It is far and away the best book I have read for anyone interested in applying behavioural and cognitive methods to couples and families (surely an area that no competent therapist can afford to ignore). In addition both consultant and trainee psychiatrists will be rewarded by dipping into the book, for they will discover an armamentarium of techniques and ideas that will integrate smoothly with standard psychiatric practice.

Professor Falloon has drawn on his extensive experience of the field and assembled an international team of contributors from England, America, Ireland, Australia, and Germany. The book is skilfully edited: the individual contributions avoid overlap and fit well together Most have illustrative case studies and useful diagrams or summaries highlighting central clinical and theoretical issues.

Although the book is basically a handbook, focusing on practice, there is a sound account of theory and the application of family therapy in a range of disorders. Subjects covered include anxiety disorders, obsessional disorders, depression, schizophrenia, senile dementia, and the developmentally disabled. There is an excellent chapter on the eclectic approach to family, marital and sexual therapy by Dr Michael Crowe. Links between behavioural methods and systematic family therapy are well described. Recent advances in therapy and in prevention are covered and I particularly welcomed a lively essay on the value of a family approach in primary health care settings.

This book is strongly recommended.

JOHN COBB, *Consultant Psychiatrist, Priory Hospital, London*

Relapse and Addictive Behaviour. Edited by MICHAEL GOSSOP. London: Routledge 1989. 305 pp. £29.95.

Strategies which address the problem of relapse should often be at the heart of an informed treatment approach in cases of alcohol and drug dependence First-line treatments in these conditions, including the various methods of detoxification, are frequently relatively undemanding upon workers and may be encouragingly successful for short or long periods, but sooner or later the potential for or the actuality of relapse has to be examined closely, and in the various levels of service either the willingness or else the skills to do this are too frequently lacking.

In the management of addictions, including the prevention or the management of relapse, treatments based upon various models of personal support, health education, flexible drug-prescribing programmes, attention to environmental factors, and crisis intervention go a very long way, and in the field of drug addiction the support for some of these elements as part of the emergency measures required to halt the spread of HIV and AIDS is a powerful demonstration that their efficacy is acknowledged, despite the lack to some extent of substantial empirical evidence.

Much of the research that has examined the details of the actual processes of relapse in addictions has been undertaken by psychologists and forms the solid basis for relapse-prevention strategies employed by that discipline, and to some extent adapted by other disciplines, in the clinical field, and this book is in essence a comprehensive review of that body of work. The editor adopts the arguably contrived view that gambling, overeating and even sex offending represent part of the spectrum of addictions, and so sections on relapse in these conditions are included. (It is fair to say that the extended analogy is easier to sustain in terms of psychological constructs than on physiological grounds, but if so is the term 'addiction' justified?) At any rate the author of the section on eating disorders rather embarrassingly states immediately that he does not consider his subject to be within the concept of addictions but the chapter is gamely included as it stands and is otherwise an entertaining essay. Other more expected sections cover the fields of alcohol, smoking and opiates, while strategies to promote behaviour change in intravenous drug users with reference to HIV and AIDS are reviewed separately by a team currently researching that area.

The contributions vary greatly in scope, style and in the extent to which they provide a literature review of their field, with several chapters excellent in the latter regard but the section on opiates particularly disappointing, relying heavily upon describing one already published study by the author's team. Conceptual issues are also discussed at various stages: for instance is the term 'relapse', which implies recurrence of a condition that had gone away, an appropriate one to describe what happens in addictions, and when does a small 'lapse' become a fully blown 'relapse'?

Specialists in addictions should probably have access to this book to be aware of the evidence which lies behind many of the psychological treatments in their field, and indeed to enhance their own clinical practice. Researchers studying relapse in addictions will find it a valuable reference work, and those involved in the related fields such as eating disorders may well find matters of interest in the chapters on their subject. Others will need a particular reason to buy the book, especially if they might be put off by poor-quality production at a highish price

Nicholas Seivewright, *Consultant in Drug Dependence, North-West Regional Drug Dependence Service, Prestwich Hospital, Manchester*

Families in Perpetual Crisis. By Richard Kagan and Shirley Schlosberg. London. W. W. Norton. 1989. 186 pp £13 95/$23.95.

Families living in perpetual crisis, repeating patterns over generations, coming to the attention of the whole range of welfare agencies but failing to engage anywhere, are most likely to receive a reactive, crisis-orientated service and to experience only further breakdown, loss and rejection at the hands of professionals. As pressures for treatment programmes which are cost-effective increase, these families risk being abandoned altogether.

Such families are addicted to crises, which provide a means of escaping the pain of facing repeated loss. Kagan and Schlosberg attempt to understand the pattern of crises and the meaning of repeated trauma, using a systemic approach to complement a dynamic understanding of loss and separation. They respect and address a family's fear of change, attempting to slow down the processes and thus reduce suspicion and establish trust. They work to give control to parents who, functioning emotionally at the level of a two- to three-year-old child, have yet to learn how to make decisions affecting their family. They build elaborate support networks to reduce a family's isolation and terror. Working in the home where they can best engage a family, they use the tools of the family therapist, live supervision and consultation to prevent the worker becoming overwhelmed. In the context of long-term involvement they use a variety of strategies to help families face painful dilemmas and slowly to make the small changes which may enable a family to remain together. This is one aspect of their treatment programme in which preventive work is fully integrated with ways of dealing with breakdown when it occurs.

The latter part of the book examines practice issues arising from placement. It is the first part of the book, however, describing the preventive outreach work with families which offers an attempt to understand the chaos and provides a rich source of strategies for making therapeutic use of the difficult, disturbing and sometimes frightening situations in which home-based workers may find themselves.

The authors' final plea for such work to be given proper recognition on the grounds that the cost to individuals, to families and to society is otherwise too high, is both timely and encouraging to those inclined to persist with this demanding work in the current sociopolitical climate.

Rosemary Loshak, *Psychiatric Social Worker, Department of Child Psychiatry, London Hospital, Whitechapel*

Psychological Management of the Physically Ill. Edited by J. Hubert Lacey and Tom Burns. Edinburgh: Churchill Livingstone. 1989. 352 pp. £29.50.

The cover of this book states its aims as providing "the practical, clinical techniques, illustrated with many case histories, that general physicians need to treat the psychological component of physical illness"; and adds that it will be of much interest to psychiatrists and social workers. How well does it achieve these laudable aims?

The editors make no apology for the nature of the book, which is a collection of monographs written by those who are experts in their particular fields. Deliberately, no attempt has been made to edit this book into a comprehensive textbook with uniformity of style or common theoretical framework for treatment. This policy is the root of both the book's strengths and its weaknesses. While it is enlightening and a joy to read of the views and treatment regimes of the chapter authors on their subjects, anyone searching for a comprehensive textbook of liaison psychiatry might be disappointed by omissions.

Given the nature of the book, the potential reader needs to check the contents carefully. The 20 chapters cover a spread of topics across physical medicine, but certain areas, such as respiratory medicine, are omitted completely (surprising in view of the psychosomatic nature of asthma and other conditions), and some of the chapters are very selective in their coverage. For example, the chapter on diabetes mellitus by Drs Pelver and Groen excellently covers the training of diabetics and the understanding and prevention of their psychological problems, but omits any mention of the recognised and often troublesome complications of eating disorders occurring in young diabetics.

These difficulties aside, it must be said that no one could fail to appreciate and grasp the essential general principles of the psychological management of the physically ill from the authors' clear presentations in respect to their chosen topics.

Any book which attempts to reach a wide readership will inevitably have problems in spanning the gulf of knowledge and attitudes between general physicians, psychiatrists and social workers. This book is certainly well pitched for general physicians and surgeons and I hope that many can find time and motivation to read it. Chapters such as those on cardiac rehabilitation and the psychological intolerance of food are particularly well matched for the physician. In spite of this, most chapters would have value for both trained and training psychiatrists.

The case histories described throughout this book are particularly valuable. They allow the reader to appreciate that the authors are not simply 'academic' experts in their fields, but that they also have deep clinical experience. Sufficient practical details of treatment regimes are given in all chapters to allow the readers to feel confident that they themselves could replicate the therapies in clinical situations. It is my hope, in spite of the authors being mainly psychiatrists and psychologists, that physicians, surgeons and medical social workers will feel able to attempt aspects of the psychological management of their patients, and certainly be more aware of such problems.

Sadly, the price of this book is not insubstantial, and although it is an essential purchase for medical and psychiatric libraries, I am sure that many individuals will await its publication in paperback form.

N. L. HOLDEN, *Senior Lecturer/Consultant Psychiatrist, Professorial Unit, Mapperley Hospital, Nottingham*

Human Nature and Suffering. By PAUL GILBERT. London: Lawrence Erlbaum Associates. 1989. 406 pp. £24.95.

The author is a cognitive psychotherapist and he describes this book as "a personal journey . . . through sociobiology, ethology, biology, philosophy and psychology". He has "tried to advance a psychobiology of mind that is consistent with evolutionary theory".

To fulfil this ambitious objective he has created a large theory of human development, relationships and psychological disorders based on ethological and cognitive principles. He describes four 'biosocial goals' or 'competencies' which have an evolutionary basis these are "care eliciting, care giving, competitive (power) seeking and co-operating"; these lead to specific affects, cognitions and behaviours, and also to disorders associated with these four constructs. Gilbert ranges very widely in his description so that it is possible to explain practically all human behaviour by means of this model.

Any author setting out such a broad theory about human behaviour has somehow to carry the reader with him, almost as a novelist has to, and he probably does this for those whose world view is ethological and cognitive. Those coming from different conceptual positions may run up against details which irritate them, but it would be wrong to evaluate this work on the basis of detail. The holistic approach will always lay an author open to charges of superficiality by specialists who resent any outsider who wanders into what they regard as their academic pastures, but this work needs to be evaluated as a whole, not as a sum of parts.

I would like to be able to say categorically whether his theory as a whole stands up or not, whether it should be accepted or rejected, but I do not think the question can be answered that simply. His theory is plausible and consistent, and represents one way of looking at the psyche, and perhaps that is as much as anyone can ever say, since there can never be a definitive description Some people will find his approach totally convincing and deeply illuminating, others will stay with their own models yet will find many useful insights in this work.

The book is systematically laid out, but the 365 or so pages of text in rather small print are sometimes hard going, the index is weak and the text needs editing

Overall the author can be congratulated on completing successfully such an enormous task. It is a fresh and imaginative view of human nature from which everyone can learn.

GLIN BENNET, *Consultant Senior Lecturer, Department of Mental Health, University of Bristol*

Not on Your Own: the MIND Guide to Mental Health. By SALLY BURNINGHAM. London: Penguin Books. 1989. 212 pp. £3.99.

Although this book is packed with commonsense and useful information, it is not fully clear who is being addressed. Sometimes it is a distressed person, sometimes their carer. Not enough detail is presented for troubled people to understand much of their own difficulties, although they can learn where to go in order to learn more. Equally, discussions are seldom deep enough to make the book useful to mental health workers. Some conventionally psychiatric problems like alcohol and drug abuse are excluded altogether. However, a huge market remains: teachers, lawyers, Citizen's Advice Bureau workers, counsellors and councillors, whose work may involve advising those with mental illness. Every public library should also have a copy.

The book includes sections which deal with problems, professional (mainly medical) sources of help, and other sources of help and information There is also an excellent final chapter listing useful organisations.

There are some problems with the book. The difficulties and dangers of precise diagnosis (e.g. with schizophrenia and dementia) are largely bypassed, and discussions of the general practitioner's role fail to underline the degree of variability of this around the country. Despite these quibbles, the book should prove helpful to many people if it is made available in an appropriate way

BERNARD INEICHEN, *Senior Research Fellow, Newham Health Authority, London*

Mild Head Injury. Edited by HARVEY S. LEVIN, HOWARD M. EISENBERG and ARTHUR L BENTON. New York: Oxford University Press. 1989 288 pp. £32.

This volume consists of 18 chapters written by 36 contributors, many of whom are neurosurgeons. The book is arranged in six sections: "History and epidemiology"; "Experimental models and neuropathology"; "Clinical management", "Neurophysiology and neuroimaging"; "Neuropsychological sequelae, postconcussion symptoms"; and "Disability and rehabilitation".

The first section will be of some interest to psychiatrists. It describes the historical development of the concept of delayed consequences of apparently mild head injury and points out the relationship between interest in head injury and the development of railway transportation and associated collisions, the increase of industrial employment, and fiscal and financial changes which allowed for insurance claims. There is also a useful account of the epidemiology of head injury, emphasising the frequent nature of mild injuries of this type and their very substantial cost in terms of treatment and possible long-term disability.

The next two sections, however, are intended for experimental neurophysiologists and neurosurgeons. Few psychiatrists are likely to wish to learn about cat fluid-percussion models of traumatic brain injury and similar techniques. The accounts of clinical and neurosurgical management of minor head injury are a detailed guide for accident department staff and neurosurgeons who are directly involved in making decisions about which patients with apparent minor head injury to admit or investigate and which to allow to go home This can only be of limited interest to those who do not work in this capacity, although the reminder that psychiatrists are far from the only clinicians who may be required to predict the likelihood of tragic and infrequent events on the basis of inadequate evidence is worthwhile.

The last three sections, however, are likely to be of much more practical value to psychiatrists and they contain a good deal of relevant information. I was particularly interested in the sections on the epidemiology and long-term consequences of head injury in children, but the account of the complex relationships between litigation and long-term symptoms in Belfast patients also raises many fascinating questions.

Although there are substantial parts of this book which do not directly concern psychiatrists, there are other parts that are of direct interest to any doctor seeing patients who may have suffered brain injury, presented in a style which will encourage the reader to take a new look at these problems. In my opinion this is a worthwhile purchase, at least for those with a reasonably comfortable budget.

EVE C. JOHNSTONE, *Professor of Psychiatry, University of Edinburgh*

Psychiatric Diagnosis, (4th edn). By DONALD W GOODWIN and SAMUEL B. GUZE. Oxford: Oxford University Press. 1989. xiv+332 pp £12.95.

This is the fourth edition and the 16th year in print of this book It is a concise, general textbook of psychiatry by distinguished American authors who consider the first edition to have been a "forerunner of DSM-III". Perhaps they are justified since a sizeable proportion of the DSM-III task force were trained in their old department at Washington University, St Louis. While the book has a strong emphasis on diagnosis and criteria, it attempts much more than this Disorders are dealt with under subheadings of definition, historical background, epidemiology, clinical picture, natural history, complications, differential diagnosis and management. Throughout, there is strong emphasis on proof for any assertions made, with practically every statement referenced in the text.

The space devoted to topics seems proportional to their relevance and topicality and the authors approach is logical and scientific. Thus, for example, the benzodiazepine problem is dealt with briefly but in a balanced way.

While *Psychiatric Diagnosis* is written from an American viewpoint it does not deal with legal or other local aspects of diagnosis and thus travels well. Overall, for such a small textbook it achieves an excellent standard and could be recommended as a short, introductory book on clinical psychiatry.

R. J. DALY, *Professor of Psychiatry, University College, Cork, Ireland*

Dictionary of Abbreviations in Medical Sciences. By Rolfh Heister. Berlin: Springer-Verlag. 1989. 280 pp. Dm58

Abbreviations are undoubtedly useful, and undoubtedly liked by authors; this is reflected in their proliferation The position of the reader, especially the novice to any particular field, is more questionable, and a book of this nature, listing over 15 000 abbreviations, is welcome, perhaps as a note of caution: casual inspection revealed that there are nine entries for NA, 14 for AD, and 23 for PM.

This dictionary does contain some abbreviations for rating scales, although PSE is not mentioned and the HRSD is confusingly listed under HRS and HPRSD. MDP has three entries, none of which is for manic-depressive psychosis, and TLE is only thin-layer electrophoresis, nothing to do with epilepsy, although the clinician is unlikely to believe that worrying side-effects might be part of normal mouse serum.

I would not recommend this book to psychiatrists. Ballière publish a handy-sized paperback book of abbreviations, much more easily thumbed through and cheaper. Heister's hardback book is up to date as of 1988 and has a pleasant design. It may well be of interest to general libraries.

Ralph Footring, *Scientific Editor, British Journal of Psychiatry*

Instinctual Stimulation of Children. From Common Practice to Child Abuse. (Vol. 1: Clinical Findings. Vol. 2: Clinical Cases). By John L. Weil. Madison Connecticut: International Universities Press. 1989. 270 pp and 460 pp. $30.00 and $45.00.

These two volumes report an investigation of children's symptomatic responses to various types of adult "erogenous, erotic, and punitive contacts". It does not only include confirmed cases of child abuse, but also looks at a range of children who the author, a child psychiatrist from Harvard, regards as "excessively stimulated" as a result of forms of parent–child interaction which vary from "common practice" to "socially unacceptable". By investigating such "erogenous and punitive contacts", irrespective of whether they are intentional, and regardless of whether they are officially labelled as abusive, Weil hopes to contribute to the scientific study of the diagnosis, treatment and prevention of child abuse.

In his 100 clinical case study, carried out over the past 25 years, Weil looks at the symptoms and dream content of children and adults who have been exposed (when young) to what he calls "various forms of instinctual stimulation". He describes 100 clinical case histories, 23 of which were obtained from the classical psychoanalytic literature (Freud, Fenichel, Rosenblatt, Spiegel, etc.) and the rest (77) of which were selected from the author's own diagnostic, supervisory, and therapeutic cases. Many of the latter are based on information from the child (or an adult recounting childhood experiences) as well as from the child's parents. There is no control group of non-clinic attenders.

Weil's own psychoanalytic orientation is never in doubt and clearly affects the way he presents and interprets the material. Both the conceptual framework and terminology are purist Freudian and clearly influence his operational definitions and observational procedures. Under "erogeneous or erotic contacts" he lists, among others, "child shares a bed with adult for the purpose of inducing child's sleep", while "adult genital erogenous contacts" appear to include "child observes adults dressing and undressing". "Excretory contacts" and thus(!) "faecal sources of stimulation" range from "child observes an adult on the toilet in the process of defaecating" through "child age five or older still wiped by adult" to "adult preoccupied with child's bowel movements" (Vol. 1, pp. 14, 15).

Volume 1 contains the 'clinical findings' which are not always clearly presented. There is, however, a useful chapter on therapeutic techniques which outlines measures directed both at child and parents. Here, some of Weil's vast clinical experience is helpful, even if one does not otherwise share his theoretical stance. This volume also contains the sample of a questionnaire designed to elicit from parents information about children's exposure to physical and sexual contacts.

Volume 2 contains the actual case studies, which unfortunately are no more than clinical vignettes, heavily edited and with little distinction between commentary and pure description They are also spiced with verbatim quotes obviously selected to underline certain points. "This picture actually turned out to portray a yellow coloured penis squirting near a brown hole, the anus, while the hand was touching brown faeces" (vol 2, p. 428): at no point is it clear whether this was the explanation the child gave or indeed the clinician's interpretation of the picture. What is missing are verbatim transcripts of interviews or sessions which would allow the reader to evaluate critically the author's conclusions. In the event, this study does not achieve its stated aims and this makes it of rather limited use despite its ample material.

K. Asen, *Consultant Child Psychiatrist, Child and Family Psychiatry Service, Barnet*

Dyslexia and Hyperlexia: Diagnosis and Management of Development Reading Disabilities. By P. G Aaron. Dordrecht: Kleiver Academic Publishers. 302 pp. £42.00.

This substantial American book is concerned with reading problems looked at from a psychological and remedial education perspective. Despite the mention of diagnosis and management in the title, psychiatric aspects of the subject are not dealt with. The point is made that reading backwardness in young people, who are intelligent and not educationally deprived in any way, is a word recognition problem and not one of difficulties in language comprehension. As well as reading backwardness, the less important condition characterised by extreme ease of reading and relatively poor comprehension is described. In addition, much research work is outlined although there is no attempt to provide a complete coverage of the relevant literature.

There is no doubt that the book contains much that is interesting and would be invaluable to anyone wishing to study reading backwardness in any detail. However, it is difficult to decipher because of the style in which it is written and the use of jargon. It is a book for the specialist reference library.

IAN BERG, *Consultant in Child & Adolescent Psychiatry, The General Infirmary at Leeds, Leeds*

Atlas of Electroencephalography. By ALAN GUBERMAN and MADELEINE COUTURE. London: Churchill Livingstone. 1989. 336 pp. £110.00.

A well produced atlas is a welcome addition to the book shelf, especially with the excellent reproduction of the tracings found here. Many are of about 20 seconds duration but, to show the evolution of seizure patterns, others are three times this length. There is a very brief introduction and the remainder is illustrations, each with the age and diagnosis of the subject, and with a reference to the source of further information on the phenomena shown. These are grouped into nine chapters, ranging from the tracings of normals, awake and asleep (including sleep stages), to those of subjects with various medical and neurological conditions. Epilepsy (ictal, interictal and status) occupies approximately half the book, indicating the place of conventional electroencephalography (EEG) in respect of seizure disorder. Coverage of paediatric work is scanty but excused by the preface, which suggests reference to appropriate texts. There is no attempt to present the changes with aging. Also, no psychiatric section is given, although some relevant illustrations are provided. Apart from a list of references specifically for each illustration, there is a bibliography. The very limited index makes quick reference difficult.

The main question in the mind of the reviewer is: to whom is this book directed? The brief preface does not indicate clearly and this does not seem to have been very carefully considered by the authors, since the phenomena illustrated are not always suitably annotated for the beginner, and for the more advanced reader, the selection of topics is not comprehensive enough in, for example, the coma section. Also, the price of over £100 would preclude many individual clinical neurophysiologists from purchasing the book, while the non-specialist in the reviewer's experience are more interested in the significance of what is shown and how often they occur in normals than in the actual phenomena themselves. Nevertheless, the volume could form a useful function, particularly for those with some experience who wish to check their knowledge. Recommended therefore with some reservations.

D. F. SCOTT, *Consultant in Clinical Neurophysiology, Section of Neurological Sciences, London Hospital, London*

Psychopharmacology: an Introduction. By RENE SPIEGEL. Chichester: John Wiley & Sons. 1989. 275 pp. £32.00.

This is the second edition of an introductory text to psychopharmacology, first published in 1981. It is aimed at the wider readership of psychologists, psychiatrists and other professionals involved in the treatment of the mentally ill, with emphasis on the clinical and psychological aspects of psychopharmacology. The book starts with an introductory chapter on modern psychopharmaceuticals, their uses and side-effects. Psychiatric nosology *vis-a-vis* pharmacotherapy is discussed, drawing solely on the Basle approach, without reference to the substantive experimental studies. Moreover, the list of drugs mentioned is not up-to-date: selective 5-hydroxytryptamine (5-HT) uptake inhibitors were not mentioned, for instance.

Two unique features of the book are chapters on the history of psychopharmacology and the effects of psychopharmaceuticals in healthy subjects, with emphasis on methodology of clinical trials. Other chapters deal with more specific methods such as neurophysiological methods and their applications, and are targeted to the specialist reader. Pharmacokinetics and pharmacodynamics are briefly covered in keeping with the aim of the book. There is a chapter on the psychopharmacology of cognition, an excellent up-to-date review of this fast-developing area. A chapter on predictors of response highlights the most promising clinical and biological predictors. The final chapter deals with psychopharmacology in the wider context of therapeutic practice. Long-term management issues are briefly covered, while use of drug therapy in combination with psychotherapy is extensively reviewed and discussed. This chapter appears to be aimed at the psychologist.

The book was written by a psychologist working in the clinic research department, Sandoz. It appears to be principally aimed at fellow psychologists with an interest in psychopharmaceuticals. The text reads clearly and is generally free of technical jargon. Trainee psychiatrists should also find it a useful introductory text to psychopharmacology, with unique features on the effects of psychopharmaceuticals in healthy subjects, methods of psychopharmacology and its history.

M T. ABOU-SALAH, *Senior Lecturer, Department of Psychiatry, University of Liverpool, Liverpool*

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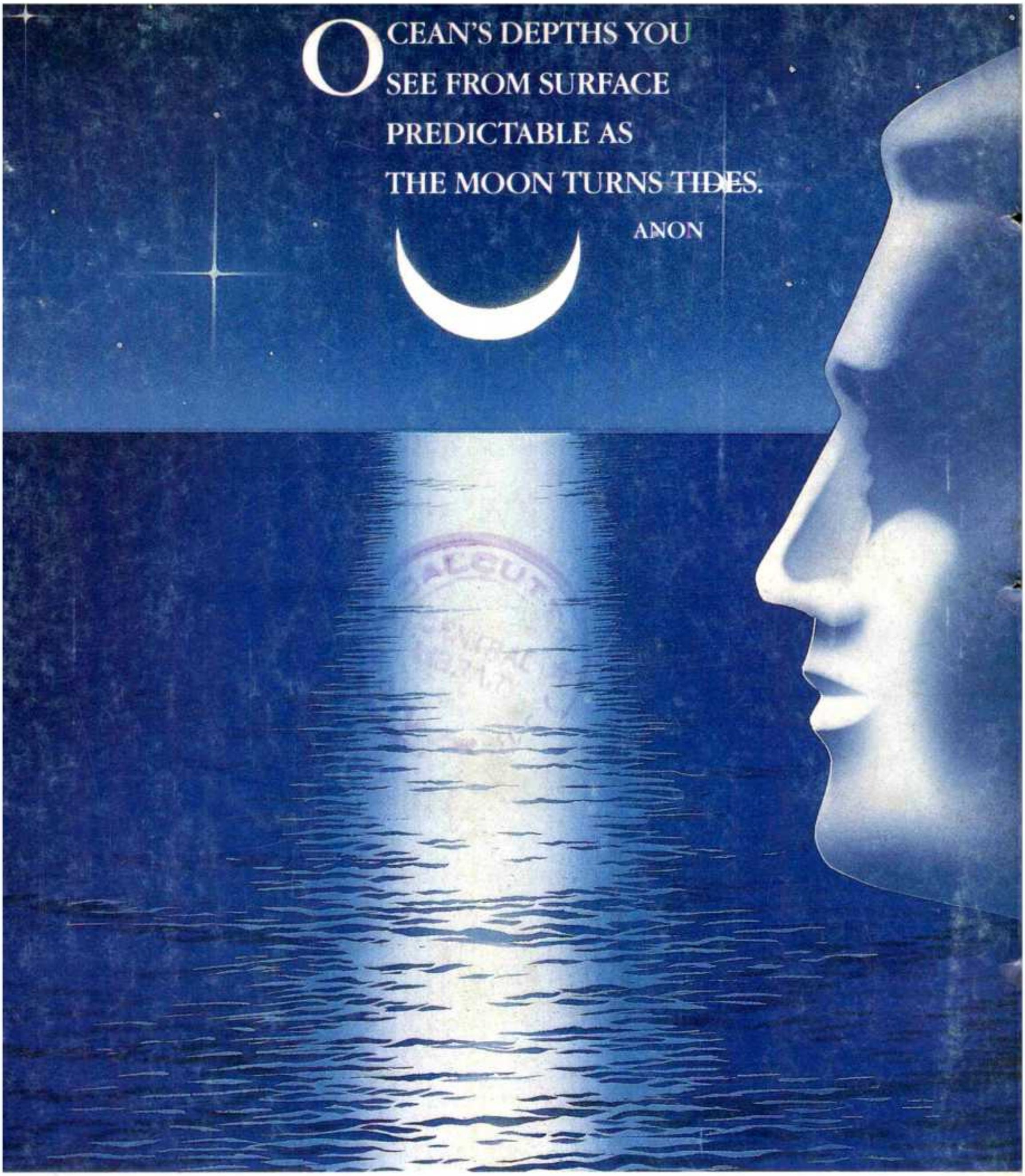

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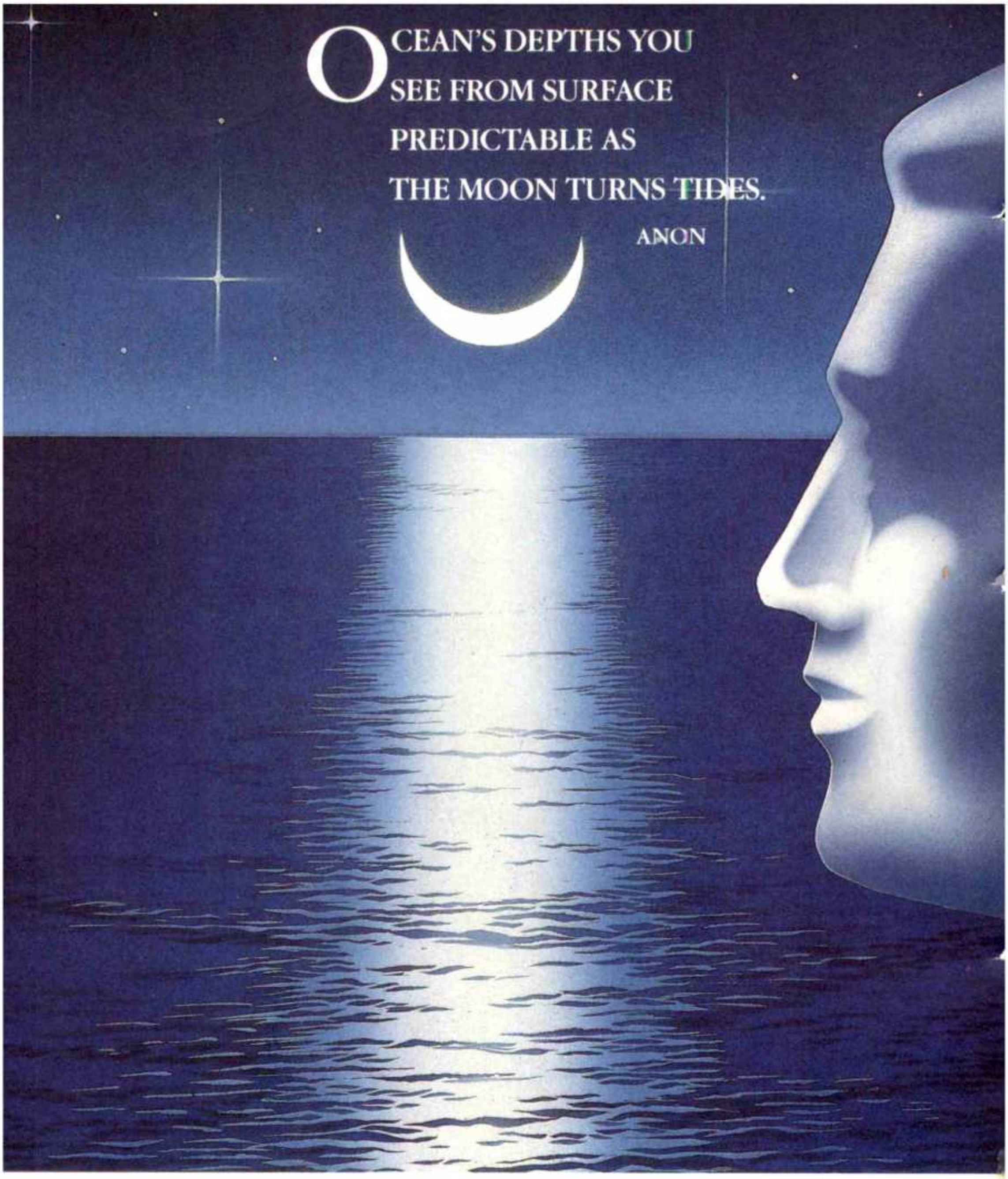

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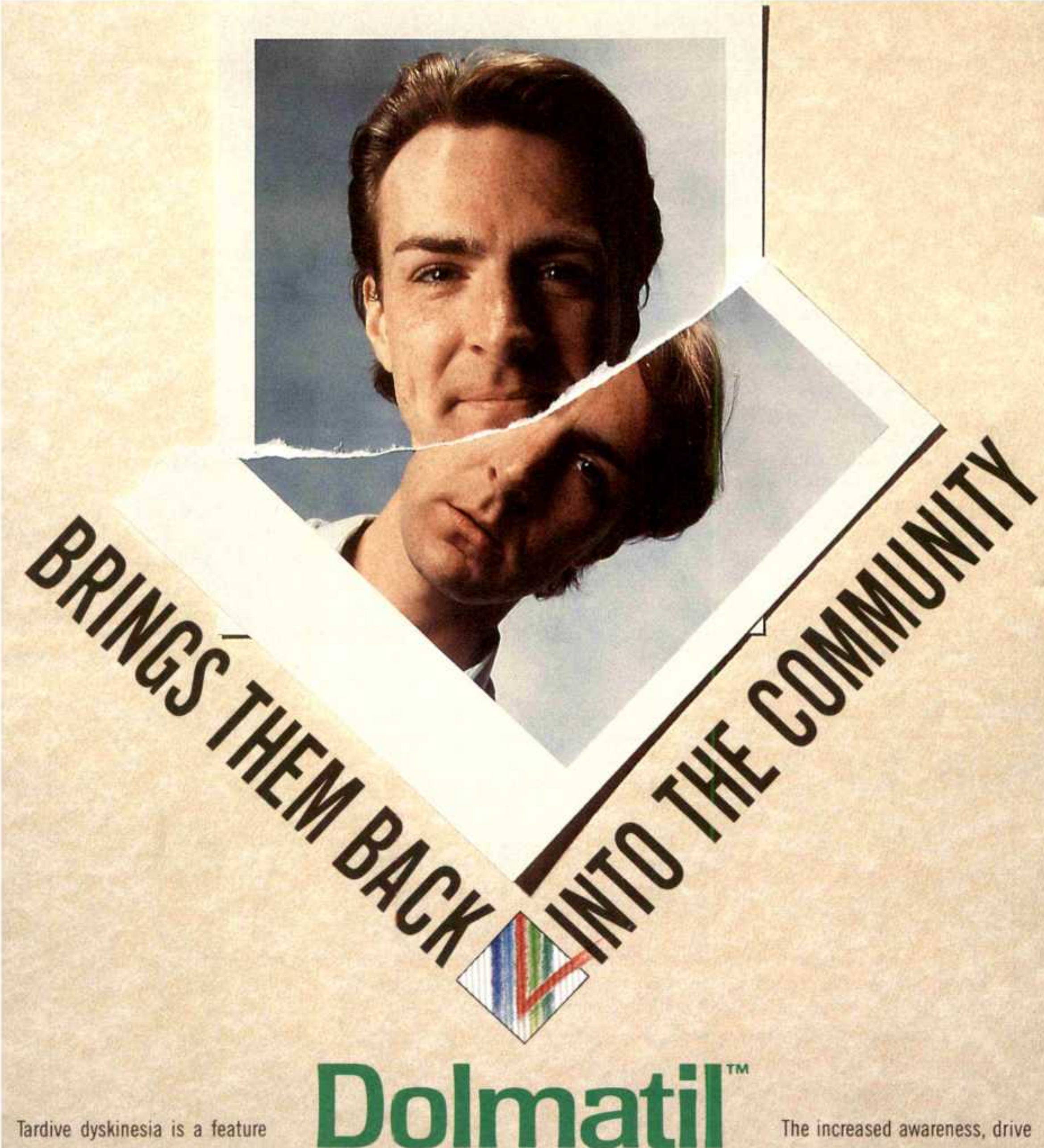
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Annotation

Sight and Insight: Regional Cerebral Metabolic Activity in Schizophrenia Visualised by Positron Emission Tomography, and Competing Neurodevelopmental Perspectives

JOHN L. WADDINGTON

Studies of cerebral metabolic activity

Over the past several years there has emerged a family of highly sophisticated but technically complex procedures for the visualisation of a range of cerebral functions in living man (Andreasen, 1988). The images they produce are so beguiling not just because of their potential to give new insights into the pathophysiology and treatment of major psychiatric disorders, but because they convey information through a quite fundamental modality: people are only convinced by what they can see. However, initial applications of such new technology have appeared just as likely to generate new questions and contradictions as to provide answers to current issues. This is readily illustrated by recent studies on the imaging of brain dopamine receptors in schizophrenic patients by positron emission tomography (PET) (see Waddington, 1989*a*).

Such technology can also be applied to study other aspects of cerebral function. As glucose is the brain's principal source of energy for metabolic and certain neurophysiological processes, there is an intimate relationship between neural activity, cerebral blood flow and glucose consumption; therefore, regional neural activity and blood flow should be reflected in the distribution of rates of utilisation of glucose. If a positron-emitting radionuclide is incorporated not into a receptor ligand but rather into a glucose analogue such as [^{18}F]-fluorodeoxyglucose or [^{11}C]-deoxyglucose, this is phosphorylated in a manner analogous to glucose but is not subject to further metabolism in the glycolytic pathway. The imaging and quantification by PET of the accumulation of this radiolabel, in terms of regional cerebral metabolic rate for glucose, is held to reflect regional cerebral 'activity'.

In initial PET studies of this type, under resting conditions or during passive somatosensory stimulation, a decreasing gradient of cerebral activity was encountered between anterior and posterior regions; this normal anteroposterior gradient was found to be attenuated in patients with schizophrenia, the deficit being termed 'relative hypofrontality' (Buchsbaum *et al*, 1982, 1984; Farkas *et al*, 1984; Wolkin *et al*, 1985, 1988; Volkow *et al*, 1987). Somewhat similar findings have been noted in bipolar affective disorder (Buchsbaum *et al*, 1986; but see Schwartz *et al*, 1987) and in depression (Baxter *et al*, 1989). While systematic searches for clinical correlates of such hypofrontality in schizophrenia during passive somatosensory stimulation have proved negative (DeLisi *et al*, 1985*a*), accentuation of hypofrontality during the performance of a frontal-cortex-dependent eye-tracking task has been reported to be more marked for patients who were more severely ill and showed the more evident negative symptoms (Volkow *et al*, 1987).

However, it must be noted that several studies, using similar PET procedures, have failed to find hypofrontality in schizophrenia (Kling *et al*, 1986; Volkow *et al*, 1986; Gur *et al*, 1987*a*); indeed, one of the most recent studies has reported schizophrenic patients to show frontal *hyper*activity (Szechtman *et al*, 1988). What sorts of factors might contribute to such inconsistencies? Were they not to have their basis in demographic and clinical differences between patient populations or in technical differences between PET procedures, the varying influence of neuroleptic medication is clearly a primary candidate. Several studies have attempted to circumvent this potential problem by studying patients who have been withdrawn from neuroleptic drugs for as little as seven days or as long as six months or more. This approach is limited by an expanding body of evidence that the persistence of both oral and depot neuroleptics may have been seriously underestimated, especially after discontinuation of long-term therapy (Wistedt *et al*, 1981; Waddington & Gamble, 1981; Gitlin *et al*, 1988; Cohen *et al*, 1988). As a corollary, the significance of studies in which the effects of neuroleptics have been examined by rescanning previously drug-withdrawn patients, after re-introduction of such medication, is unclear; both a generalised increase in cortical and subcortical deoxyglucose utilisation, without any effect on anteroposterior gradient (DeLisi *et al*, 1985*b*; Wolkin *et al*, 1985; Buchsbaum *et al*, 1987), and negative findings (Gur *et al*, *1987b*) have been reported. During the performance of an auditory discrimination task believed to place demand on frontal cortex, pre-frontal cerebral activity was found to be reduced in drug-withdrawn schizophrenic patients in a manner unrelated to performance deficits; patients receiving neuroleptics, however, appeared to show both an attenuation of this deficit in deoxyglucose utilisation and some restoration of the normal relationship between higher frontal utilisation and greater accuracy of performance (Cohen *et al*, 1987, 1988).

The issue of a potential confounding effect of medication is, of course, best addressed by performing PET studies of deoxyglucose utilisation in drug-naïve patients, in a manner similar to PET studies of dopamine receptors (see Waddington, 1989*a*). In the first such study, of three drug-naïve patients and a fourth who had received only three months of treatment a year previously, no metabolic hypofrontality was found; indeed, metabolic activity tended to be higher in several regions, including the frontal area, and this appeared to be accentuated after acute neuroleptic challenge, especially in the basal ganglia (Volkow *et al*, 1986). In the most recent study (Szechtman *et al*, 1988), eight never-medicated, acute schizophrenic patients showed a pattern of enhanced activity in the frontal area and diminished activity in the posterior cortical area, and this pattern was qualitatively similar in patient populations scanned after one and 4–14 years of neuroleptic medication; while differences from control values were less marked the more prolonged the treatment period, duration of treatment is clearly confounded with duration of illness. Additional PET studies of deoxyglucose utilisation in drug-naïve patients are urgently required, to support or contradict these controversial findings, and are in progress (Buchsbaum *et al*, 1989). Two other PET studies of drug-naïve or predominantly drug-naïve schizophrenic patients, utilising inhalation of ^{15}O and $[^{15}O]CO_2$ or intravenous injection of $[^{15}O]H_2O$, found neither regional cerebral metabolic utilisation of oxygen nor cerebral blood flow to show hypofrontality (Sheppard *et al*, 1983; Early *et al*, 1987). In the latter study, there was a particularly prominent and reproducible elevation of blood flow in the left globus pallidus; previous incidental findings in PET studies of deoxyglucose utilisation have included abnormalities in basal ganglia activity, in addition to abnormalities in laterality and cortical–subcortical gradient, and may include hyperutilisation in the temporal lobe (DeLisi *et al*, 1989).

Are there other potential sources of systematic error when comparing schizophrenic patients with normal subjects in such PET studies? There is evidence that PET estimates of deoxyglucose utilisation can be influenced by structural brain changes (Yoshii *et al*, 1988), and there is now a substantial, though not entirely consistent, body of studies which indicate structural brain pathology in schizophrenia (Shelton *et al*, 1988; Waddington, 1989*b*); these factors might contribute to variations between PET studies. Some investigators have attempted to control for such an effect by comparing PET values between patients with and without anatomical brain changes as identified by a structural imaging procedure, usually X-ray computerised tomography (CT). Most recently, Yoshii *et al* (1988) have found differences in cerebral morphology among healthy volunteers, as identified by magnetic resonance imaging (MRI), to exert a significant effect on glucose utilisation; algorithms for the correction of such regional PET data to units of actual neural tissue, via additional MRI procedures, are available (Videen *et al*, 1988).

Important as this is, Yoshii *et al* argue that the state of behavioural arousal may make an even greater contribution to variance in deoxyglucose utilisation between human PET studies, and suggest that such studies should be conducted under conditions of defined behavioural activation rather than in the resting state. A contrary view would be that studies of patients under resting conditions might best reflect the natural state of schizophrenia, unmodified by artificially imposed demands. It should not be overlooked that PET is still a new, highly complex and continually evolving technology; there remain a very large number of experimental factors that may potentially lead to artefactual data, and these make it difficult to compare directly individual clinical studies (Holcomb *et al*, 1989).

Where do all these considerations of PET procedures leave the notion of hypofrontality in schizophrenia? As Weinberger (1988) reminds us, Kraepelin himself speculated that dementia praecox might involve the frontal lobes, and there is considerable (but not conclusive) evidence for reduced frontal cerebral blood flow in schizophrenia using the alternative ^{133}Xe inhalation technique. In the most sophisticated studies of this type, Weinberger *et al* (1986) reported reduced blood flow in the dorsolateral pre-frontal cortex of drug-withdrawn schizophrenic patients during performance of the Wisconsin Card Sort Test, but not during a variety of other neuropsychological test procedures; this suggests that frontal dysfunction may be regionally specific, and most evident when demand is placed on this pre-frontal area through neuropsychological tests. The more recent studies are particularly provocative because they additionally indicate an association of such pre-frontal dysfunction with lateral ventricular enlargement (Berman *et al*, 1987) and abnormalities of central dopamine metabolism (Weinberger *et al*, 1988); this suggests the possibility of integrating (task-related) hypofrontality in schizophrenia with both structurally based theories and a variant of the dopamine hypothesis.

Neurodevelopmental theories

These ideas were incorporated into a neurodevelopmental schema that sought to account for the typical emergence of diagnostic symptoms in the late teens. It did so in terms of a late functional maturation of certain pre-frontal systems putatively affected much earlier in the course of cerebral development (Weinberger, 1987). However, any schema which purports to account for so wide a variety of experimental findings, and to integrate such diverse pathophysiological theories, might engender almost as much curiosity as one which stands resolutely in isolation and can accommodate none of these. Also, the evidence for abnormalities of frontal structure in schizophrenia does not so far appear to be either extensive or consistent (Shelton *et al*, 1988; Suddath *et al*, 1989; Andreasen *et al*, 1989) and recent neuropathological studies point more reliably (Lantos, 1988) to abnormalities in the temporal lobe. It may be that frontal dysfunction results, at least in part, from structural abnormalities in regions such as the temporal lobe, which communicate with the frontal cortex (Suddath *et al*, 1989).

Can PET studies of frontal lobe function in schizophrenia also be compatible with current dopaminergic and neurodevelopmental perspectives of schizophrenia (Waddington, 1988)? There is some evidence that the effects on regional deoxyglucose metabolism of dopamine antagonist neuroleptics and of the indirect dopaminergic agonist amphetamine appear to be opposite, at least in the basal ganglia of neuroleptic-withdrawn schizophrenic patients (Buchsbaum *et al*, 1987; Wolkin *et al*, 1987). However, this is not the same as relating measures of deoxyglucose utilisation by PET to indices of dopaminergic function, either in the frontal cortex or elsewhere. The only PET study of deoxyglucose metabolism that has been systematically interpreted in neurodevelopmental terms is that of Szechtman *et al* (1988); this relates not to hypofrontality but to their finding of resting *hyper*frontality in neuroleptic-naïve, acute schizophrenic patients, and the effects thereon of long-term neuroleptic administration. They suggest that these results might indicate frontal hyperinnervation, in accordance with the hypothesis of impaired late synaptic elimination in schizophrenia, as it is well established that optimal neuronal interconnectivity is normally attained through processes which involve the programmed elimination of aberrant and redundant neuronal connections (Feinberg, 1982; Murray & Lewis, 1987). Schizophrenic and some affective patients have been reported (Jorgensen *et al*, 1984; Lyons *et al*, 1988; Jorgensen, 1988) to show elevated plasma levels of a neural cell adhesion molecule (NCAM), a morphoregulator capable of detailing the synaptic connections that are critical to the development of neuronal function in the central nervous system. Such increases in serum NCAM levels in schizophrenia might be consistent with an elevation of synaptic turnover rate associated with frontal hyperinnervation. However, these notions must be set against those discussed above, particularly the emerging body of neuropathological and neuroimaging studies which point to abnormalities of temporal, rather than frontal, structure that may disregulate frontal function. These temporal abnormalities have themselves been interpreted in terms of neurodevelopmental anomaly rather than neurodegenerative disease (Lantos, 1988).

It seems almost inevitable that initial studies, utilising such complex but potentially very powerful procedures in relatively small numbers of patients, will throw up inconsistencies. The data of Szechtman *et al* might be reconciled with those of Weinberger and associates by recognising that these studies differ not just in technique (deoxyglucose utilisation by PET v. regional cerebral blood flow by the ^{133}Xe technique), but also by patient characteristics (acute v. more chronic), medication status (drug-naïve v. neuroleptic-withdrawn), and test conditions (resting v. task). Clearly, the primary need is for more extensive studies with these techniques and test paradigms in acute, neuroleptic-naïve schizophrenic patients, in comparison not just with normal subjects but with patients having affective and other disorders. Nevertheless, it remains curious that both of the studies considered above appear consistent with (distinct) variants of neuronal dysplasia and thus, in different ways, extend current notions of schizophrenia as a neurodevelopmental disorder. Can they both be right?

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Review Article

Dystonia – A Potential Psychiatric Pitfall

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The manifold presentations of dystonia have been acknowledged as a spectrum of illness. Dystonia is primarily idiopathic or drug-induced; purely psychogenic causes would seem to be rare, although such an aetiology is commonly assumed. Response to medication in idiopathic cases is unpredictable, although anticholinergic medication and behavioural programmes are being evaluated.

It may be true to say that at present general psychiatrists come into contact with disorders of movement as often as general neurologists. Despite a great deal of interest in the subject of movement disorders in the psychiatric literature in recent years, there is evidence that in routine clinical practice a considerable amount of abnormality of this type is overlooked (Weiden *et al*, 1987). Psychiatrists still seem unattuned to seeking out movement disorder, especially if mild, and perhaps equally important, may feel themselves ill-equipped to categorise such essentially neurological abnormality. In view of the prevalence and potential significance of these features, oversight in this area, whatever its basis, represents a major limitation to comprehensive patient assessment.

While hyperkinetic disorder of choreoathetoid type has been extensively described in the psychiatric literature, relatively little has been written about dystonia, a concept which has itself undergone refinement by neurologists in recent years.

History

The term 'dystonia' has a varied history. Some authors applied it in what might be called a generic sense. Thus, for Wilson (1940) dystonia meant "any variability in muscle tone," while Denny-Brown (1962) emphasised the "fixed abnormalities of posture" that result from persistent tonal changes, as is to be found for example in hemiplegia or Parkinsonism. This 'generic' usage has largely been abandoned, and dystonia is now usually reserved for either a specific syndrome or, increasingly, for the abnormalities characteristic of this syndrome.

Hammond (1871) was the first to describe movements having the major characteristics of this type of disorder – namely gross muscle spasms and resulting distortions of normal posture. In his two patients the limbs were affected resulting in an *inability to hold a fixed position*, a sign he referred to as 'athetosis'. Athetosis and dystonic abnormality differ only in distribution but not essentially in quality. Thus 'athetosis' is strictly 'distal dystonia' (Marsden, 1976*a*) – or, confusingly, dystonia has been viewed, in Wilson's (1940) terminology, as 'proximal athetosis'!

Schwalbe (1908), in his doctoral thesis, described a syndrome of 'chronic cramp' in three siblings of a Lithuanian Jewish family which, being a psychiatrist, he attributed to psychological (i.e. hysterical) causation. Three years later, however, the neurologist Oppenheim described six new cases and asserted that the basis of the disorder was not psychological but a variability of muscle tone with consequent distortion of normal posture. To reflect this he suggested the name by which the condition is still known, 'dystonia musculorum deformans' (Oppenheim, 1911). Since then neurologists have frequently used the term 'dystonia' as a sort of short-hand for this uncommon, often familial disorder. Other early workers on the subject chose to emphasise that the characteristic movements of this condition had a sustained and typical twisting quality (Ziehen, 1911; Flatau & Sterling, 1911), a view accommodated in the alternative name of 'torsion spasm', also still widely used.

Dystonia musculorum deformans (DMD) is a rare disorder. It has however been recognised for many years that involuntary movements with the same sustained, twisting characteristics as those typical of DMD could affect isolated muscle groups or local body parts. These abnormalities tend to have a later age of onset and rarely generalise to involve the whole body. The names traditionally applied to these conditions reflect their local distributions, such as blepharospasm, torticollis, or writers' cramp. In addition to their descriptions in neurological texts, these conditions have, interestingly, also figured heavily in the pages of psychiatric textbooks (e.g.

Abse, 1966) as examples of conversion hysteria, illustrating the parallel but separate paths the two specialities have often taken in the past century.

In the last few years there has been a reappraisal of the position of localised dyskinesias of this type in relation to the more generalised form of disorder. This has resulted in a unifying classificatory system encompassing all the dystonias.

Definition

In a condition with such a varied history, producing a unified definition is not easy. Early on Wechsler & Brock (1922) emphasised two main clinical forms which they referred to as 'myostatic' and 'kinetic'. Since then some authors have emphasised the static elements of the disorder in terms of the attitudes produced as a result of the abnormal muscle contractions, while for others the dynamic aspect of the abnormal contractions themselves was more important (for review of definitions see Fahn *et al* (1987)).

In 1984, an *ad hoc* committee of members of the Scientific Advisory Board of the Dystonia Medical Research Foundation produced the following definition:

> "Dystonia is a syndrome dominated by sustained muscle contractions, frequently causing twisting and repetitive movements, or abnormal postures." (Fahn *et al*, 1987)

Classification

Dystonias have been classified by age of onset, by cause, or by distribution (Table I). Classification by age of onset is valuable because of the prognostic implications. Age of onset is probably the most important factor with regard to severity and the likelihood of generalisation (Marsden *et al*, 1976; Cooper *et al*, 1976; Fahn, 1986). Thus, the younger the onset (especially under ten years of age) the more likely is generalisation to take place. By contrast generalisation is rare with onset after 40 years (Marsden *et al*, 1976; Fahn *et al*, 1987). Psychiatrists are most likely to come in contact with adult-onset cases, so classification by cause or distribution is more useful to their practice.

TABLE I
Proposed classification of dystonia

Classification by:	*Criteria*
Age of onset	< 12 years of age 13–20 years of age > 20 years of age
Aetiology	Idiopathic (a) sporadic (b) familial Symptomatic
Clinical distribution	Focal Segmental Multifocal Generalised Hemidystonia

After Fahn *et al* (1987).

While most dystonias are idiopathic, symptomatic cases associated with neuroleptic drug treatment are those most frequently encountered in psychiatric patients. It is encumbent on all clinicians however not to be blinkered in their approach to these abnormalities as many other causes of symptomatic dystonia have been reported apart from neuroleptic drug exposure. The reader is referred to the exhaustive differential diagnosis presented by Fahn *et al* (1987) for details, where over 40 associated disorders are listed. Although most of these are clearly neurological (and some very rare), a number may be seen in psychiatric contexts. These include Wilson's disease, Huntington's disease, Lesch–Nyhan syndrome, encephalitis, head injury, intracranial tumours, multiple sclerosis, cerebrovascular accident, carbon monoxide poisoning, and exposure to centrally acting drugs other than dopamine-blocking agents (see below).

A descriptive classification based on distribution is clinically the most straightforward. This divides dystonia into five main types:

(a) focal: affecting one body area only
(b) segmental: affecting two or more contiguous body areas
(c) multifocal: affecting two or more non-contiguous body areas
(d) generalised: affecting multiple, disparate body areas
(e) hemidystonia: affecting ipsilateral limbs, with or without involvement of ipselateral trunk or face.

By this classification a number of differently named clinical entities are subsumed under recognisably similar terminology. The arguments justifying this have been presented by Marsden (1976*a*) and Fahn *et al* (1987). Thus blepharospasm, torticollis, and writers' cramp all become examples of *focal* dystonia, while Meige's syndrome (see below) becomes an example of a *segmental* dystonia.

Hemidystonia is usually symptomatic (Fahn *et al*, 1987), secondary to a contralateral brain lesion, especially of caudate/lenticular nucleus/thalamus (Marsden *et al*, 1985). It must be remembered however that movement disorder associated with neuroleptic exposure can present unilaterally (Waziri, 1980; Myslobodsky *et al*, 1984; Wilson *et al*, 1984), although usually the abnormality is then restricted to one limb.

General clinical characteristics

Dystonia has two components: the active or 'kinetic' movements themselves (the dyskinesias), and the maintained postural abnormalities or persistent attitudes resulting from abnormal muscular contraction. These may occur singly or in combination. Any striated muscle can be involved, but in the non-generalised varieties, the head and neck are most commonly affected (craniocervical dystonia). Likewise, when drugs and other potentially causative factors can be ruled out, dystonia is probably the most common form of *spontaneous* craniocervical dyskinesia (Jankovic, 1988*a*).

Onset may be rapid, but usually the condition evolves slowly, over months or years. The initial complaints may be vague and non-specific, such as nuchal ache or eye irritation, or muscle tension may be evident only when the patient performs some voluntary activity in the affected area ('action dystonia'). With progression, activity in distant muscle groups may precipitate dystonia ('overflow' phenomenon). As a general rule, dystonic movements are invariably aggravated by voluntary activity, although eventually movements and abnormal postures may be evident at rest.

It has recently been appreciated that the movements of dystonia can vary greatly in the speed with which they develop. While typically they tend to be slow and sinuous, some can be rapid in onset and difficult to distinguish from other dyskinesias such as tics or chorea, and may even have the 'lightening' quality of onset of myoclonus – so-called myoclonic dystonia (Obeso *et al*, 1983). Like their slower counterparts, these rapid movements have the same twisting characteristics and tend to be continual and repetitive. It has been suggested in addition that abnormal movements should be sustained for at least one second at the point of maximum contraction to qualify as dystonic (Fahn, 1984). Other involuntary movements may however occur at the same time, a particular association being noted with postural tremor, and a family history of essential tremor may also be found (Marsden, 1976*a*).

Dystonias have unusual characteristics, some of which are shared with other extrapyramidal movement disorders, some of which are unique. Thus in common with other involuntary movements they are influenced by the degree of mental arousal, and are consequently exacerbated by anxiety or other adverse mental state features or by stress in general. Conversely they are eased by calm, relaxation or sedation, and may be suppressed completely during hypnotic states. Such psychological factors may underly a certain degree of periodicity in symptom expression in that movements can be evident on some days but not on others, and sustained postures can relax intermittently. Like most other dyskinetic states, dystonias disappear during sleep. Specific focal dystonias may exhibit specific precipiting factors (see below) and a wide range of task-specific dystonias are precipitated only by use of occupational implements of a certain type, size, or weight.

In addition, however, dystonias have certain other characteristics that are frankly bizarre. The motor action precipitating the dystonia may be highly selective, even within a specific body region, preserving other actions. Thus a patient with deforming dystonia of the legs which precludes normal forward gait may be able to walk backwards or run freely. One of the author's patients with severe oromandibular dystonia and profound scoliosis lost both his disfiguring movements and his postural deformity on the dance floor, and has taken up dancing with vigour since recognising himself the association with symptom suppression.

Many patients find, at least in the earlier stages of their disorder, that certain tactile or proprioceptive stimuli can reduce or even abolish their dystonia. These various activities 'devised' by the patient have been referred to as 'sensory tricks' or 'antagonistic gestures', and are said to be unique to dystonia and hence of diagnostic value (Fahn *et al*, 1987). Examples of this are attenuation of head deflection in torticollis by touching the chin, or holding the back of the neck with the opposite hand to the direction of rotation. One patient found that while this latter procedure did not markedly alter the degree of displacement from her severe torticollis, it did greatly ease the subjective discomfort. Another patient, in effect functionally blind with blepharospasm, could achieve a sufficient degree of eye opening to allow walking by rubbing his upper dental margin.

In view of these unusual characteristics it is easy to see how dystonias could be considered primarily psychogenic disorders.

Major clinical types

Acute dystonias

Acute dystonias are probably the commonest (*Lancet*, 1985). Although secondary to antidopaminergic medication, they are best classified separately from other symptomatic dystonias in view of their sudden onset and brisk response to treatment, which implies a distinct, or possibly more transient, pathophysiology.

It is important to remember that any drug with dopamine-blocking potential can cause these bizarre abnormalities. Thus while all classes of typical neuroleptics are undoubtedly the commonest causative agents, other drugs less frequently encountered in a psychiatric setting may be implicated, such as the antiemetic substituted benzamide metaclopramide and certain antihistamines. One recent immigrant to the UK from West Africa was referred to the author as a hysterical malcontent expressing his contempt for Western culture by permanently extruding his tongue. As a poor traveller he had taken travel-sickness pills for the flight and his 'contempt' was instantly accommodated following intravenous anticholinergic.

The distribution of acute dystonic reactions interestingly parallels that of the idiopathic dystonias in that it depends to some extent on age. Thus in children they are often severe and generalised, whereas in adults they are more usually (though not exclusively) craniocervical (Ayd, 1961; Marsden *et al*, 1975). They develop suddenly, usually within 24–48 hours of first exposure to the provoking agent, or after dose increment (Ayd, 1961), although may be delayed until 72 hours or longer with depot preparations (Johnson, 1973). They can last, untreated, from minutes to hours, and may come in runs, with periods of relative quiescence between.

The clinical features are varied and often bizarre, and can include aversive eye movements, blepharospasm, trismus, mouth opening, tongue protrusion, grimacing, distortions of lips, displacement/rotation of head and neck, hyperpronation, finger/wrist posturing, opisthotonos and other axial distortions, and gait disturbance. Dysarthria, dysphagia, jaw dislocation, tongue amputation, and respiratory stridor with cyanosis may ensue. A promonitory phase of local cramp-like discomfort or tightness of jaw or tongue may occur, making chewing, swallowing or speaking difficult, or such features may be the sole manifestation or disorder. It is crucial to view all 'lists' of acute dystonic symptoms with circumspection as none can be comprehensive in recording all the possible combinations that will be encountered clinically.

Acute dystonias with neuroleptic usage may be more frequent than is often assumed. Ayd's (1961) figure of 2.3% is still quoted, but his method of hierarchical classification of extrapyramidal reactions means that this figure refers to those of his sample who experienced only acute dystonia without subsequent akathisia or Parkinsonism. Addonizio & Alexopoulos (1988) suggested a prevalence of 25% overall; in addition they demonstrated the clear effect of age on prevalence – while 2% of elderly patients (mean age 69 years) on neuroleptics had shown dystonic reactions, the comparable figure for young patients (mean age 26 years) was 31%, a finding not attributable to differing prescribing habits. While it is generally believed that high-potency antipsychotics are most likely to be implicated, Addonizio & Alexopoulos also found that in terms of chlorpromazine equivalents, young patients who developed acute dystonias were receiving *lower* doses than those who did not, implying an element of individual susceptibility. One factor in this may be gender, as males appear to be twice as commonly affected as females (Swett, 1975; Ayd, 1961, 1983).

Acute dystonias, in addition to the frequently grotesque appearances they impart, may be painful and invariably are exceedingly distressing. Following the introduction of neuroleptics they were often misdiagnosed as tetanus (Melvin, 1962), status epilepticus, or manifestations of catastrophic CNS infection, resulting in needless investigations such as lumbar puncture and inappropriate interventions such as tracheostomy (Ayd, 1961). In the author's experience, however, they are still sometimes misdiagnosed as 'hysterical'. There is little excuse for such an error in psychiatric practitioners. Any patients with the sudden onset of subjective stiffness or typical movement and postural disorder in any body part, *especially* if the clinical appearance is bizarre, should be given a trial of parenteral treatment, whether or not an appropriate drug history can be obtained. Patients may not wish to be forthcoming on such matters, may be ignorant of what they have ingested, or may be so distressed and disabled, especially by lingual or oropharyngeal/laryngeal dystonia, that they are unable to respond to specific questions adequately.

A trial of treatment should not await resolution of the drug question. Appropriate treatments are safe, readily available and rapidly acting (within minutes). Anticholinergics are now the most widely used. Notwithstanding the above, however, the euphorogenic properties of these preparations (Shader & Greenblatt, 1971; Jellinek, 1977; Kaminer *et al*, 1982) and the pharmacological sophistication of many psychiatric patients must be borne in mind when faced with repeated requests for anticholinergics at inappropriate phases of neuroleptic exposure or in the continuing absence of objective signs. In the past barbiturates, diazepam, and the antihistamine diphenhydramine have also been reported as effective but have largely been superseded.

Oculogyric spasms

J. P. Albrecht gave the first reliable account of ocular spasms occurring in epidemic encephalitis as far back as 1695 (quoted in Jelliffe, 1929). From 1921 onwards, so-called 'oculogyric spasms' were increasingly described as a late sequela of encephalitis lethargica, although a similar phenomenon was also noted in neurosyphilis. Episodes occurred almost exclusively in the context of the Parkinsonism that followed the acute illness (Jelliffe, 1929; Wilson, 1940) and were characterised by the sudden deviation of both eyes upwards or upwards and to one side, usually the right. Spasms were maintained ('tonić') or less commonly jerky ('clonic') and were often associated with head/neck deviation ('cephalogyric spasms', usually retrocollis). They lasted from minutes to hours. Pain was sometimes noted but a particularly unnerving anxiety or fearfulness was regularly experienced (Jelliffe, 1929; Wilson, 1940). Precipitating factors included any states of extreme emotion as well as a range of physical factors such as excessive sensory stimulation (e.g. bright light, noise, heat) or fatigue and exercise (Van Bogaert, 1934). Sometimes the patient recognised 'warnings' (Wilson, 1940). A diurnal variation with increased afternoon/evening frequency was often noted, possibly related to the effects of increasing muscular fatigue.

Present-day psychiatrists would recognise some of the above as comparable with what is seen with antidopaminergic treatment. Usually this occurs as part of an acute reaction following initial exposure but the author has encountered such spasms as an isolated phenomenon usually in the course of gradual reduction of depot regimes.

The interest in this shown by a previous generation of neurologists has not been shared by present-day psychiatrists and nothing is now known of precipitating factors, 'warnings', diurnal variation, or the prevalence of associated abnormalities. This is of interest as it is clear that post-encephalitic ocular deviation was but one manifestation of an often complex neuropsychiatric disturbance (Jelliffe, 1929). Dystonia in other sites, autonomic overactivity, altered consciousness, visual misperceptions and compulsive 'forced' thinking/ruminations were all described (Jelliffe, 1929; Wilson, 1940), as were speech disturbances either premonitory to or during an attack. These included palilalia, stereotyped phrasing, echolalia, logorrhoea, mutism, and forced shouting (so-called 'klazomania') (Van Bogaert, 1934; Wohlfart *et al*, 1961). The author is unaware of work addressing all or any of these points in the context of drug causation.

When a range of other neuropsychiatric features or prominent affective disturbance is present the condition would justify being referred to as a 'crisis', but with uncomplicated eye deviation 'oculogyric spasm' or simple 'oculogyrus' would seem more appropriate.

Focal dystonias

Torticollis

Although uncommon, idiopathic spasmodic torticollis is the most prevalent of the focal dystonias (Duane, 1988). Formerly it was estimated to have accounted for 1 in 1200 psychiatric referrals (quoted by Lees, 1985). The maximum age of onset is in the fifth decade and while some authors report an equal sex prevalence (Lees, 1985), others note a female excess (Duane, 1988).

Although the sternocleidomastoid is the most commonly affected muscle, there is no single characteristic presentation. Contrary to general belief, the pattern of muscular involvement is often highly complex, which results in postural distortions encompassing every possible head position, not just the simple rotational abnormality implied in the name (Fahn, 1984). It has been pointed out that only by marked effort can normal individuals simulate these changes (Lees, 1985).

Abnormal head position is noticed first by others in about 25% of cases, and in a minority may be preceded by nuchal discomfort. Movement may initially be triggered by bimanual tasks such as driving, or by attempts to maintain a formal posture, as for example in a public gathering. Deviation can be sustained or intermittent, with progression in severity over approximately 18 months. Remissions, especially in those under 40, are not uncommon (approximately 20%), but neither is subsequent relapse (Duane, 1988).

Pain, especially at the points of insertion of the sternomastoid and upper trapezius, is common, although later secondary degenerative joint disease may be a greater source of discomfort than muscle pain.

'Sensory tricks', including touching the chin, vertex, or back of the neck with the hand contralateral to the direction of rotation, may alleviate symptoms, as may yawning, swimming, or lying down. Indeed, patients may report themselves free of dystonia for up to an hour after rising in the morning. Emotional distress, reading, writing, menstruation, pregnancy, and alcohol (especially 'the morning after') have all been reported as exacerbating influences (Duane, 1988).

In addition to head displacement, examination may reveal scoliosis (50%), palpable muscle spasms and even enlargement, other facial dystonias, and head tremor, especially of horizontal or 'negation' type.

The aetiology is in general unknown. A small number of cases appear to be familial (Gilbert, 1977) and associations have been reported with head/neck trauma (Sheehy & Marsden, 1980; Duane, 1988), diastolic hypertension, thyroid disease, especially in women, and alcoholism, especially in men (Duane, 1988). There are no predisposing personality attributes (Cockburn, 1971) and depression when present is a secondary rather than an aetiological factor (Tibbetts, 1971).

Less common variants are laterocollis (head to shoulder) and the bilateral spasms resulting in retrocollis (10% of idiopathic cases) and antero- or antecollis (5%).

Blepharospasm

Blepharospasm is characterised by intermittent or persistent involuntary closure of the eyes due to spasmodic bilateral contractions of the orbicularis oculi muscles. It usually begins in the sixth decade and there is a female preponderance (Jankovic *et al*, 1982). In the early stages it may present with non-specific discomfort or a complaint of excessive, uncontrollable blinking (blepharoclonus), and may be unilateral. It is often misdiagnosed as 'dry eyes' (Jankovic, 1988*b*). Progression leads to increasing incapacity and ultimately functional blindness.

In the early stages precipitating factors include upward or downward gaze, bright light (patients often wear dark glasses), strong direct wind, smoke and other air pollutants, fatigue, and emotional distress. Active concentration may on the other hand sustain eye opening, leading to a false impression of the degree of disability at examination (Marsden, 1986).

A wide range of 'tricks' may help, such as touching the forehead, pinching the neck, humming, or singing (Jankovic, 1988*b*). Severity may fluctuate.

In addition to forced eye closure, another clinical variant is mild to moderate tonic contraction of orbicularis oculi that results in persistent narrowing of the palpebral fissure (Tolosa & Marti, 1988). This type rarely interferes with vision but imparts a rather intimidating look to the patient. The author has seen several cases of this in patients on long-term neuroleptics where the somewhat menacing expression had been attributed to the mental state. This may be a commoner abnormality in such patients than has hitherto been appreciated.

Occasionally, once the eyes have been closed by involuntary contraction, the patient experiences difficulty in opening them again even though there is no involuntary activity opposing their efforts. This has been referred to as 'apraxia of eye-lid opening' (Goldstein & Cogan, 1965) or, perhaps more accurately, 'involuntary levator inhibition' (Lepore & Duvoisin, 1985), and has been reported as a common finding in Meige's syndrome (Tolosa & Marti, 1988).

Isolated blepharospasm is often referred to as 'essential' in type, but 80% of cases with eye signs show additional dystonic abnormality in eyebrow, facial, oromandibular, lingual, laryngopharyngeal or cervical muscles (Jankovic, 1988*b*), so the term 'essential blespharospasm' should be used with care.

Excluding local ophthalmological disorders, the cause of blepharospasm is unknown, although in psychiatric practice this is overwhelmingly related to neuroleptic drug exposure.

Writers' cramp and the occupational dystonias

Writers' cramp (scrivener's palsy, *schreibkrampf*, *crampe des ecrivains*) is probably one of the oldest known forms of dystonia (for a historical account see Sheehy & Marsden, 1982; Lees, 1985) and the one which has in the recent past been most strongly associated with psychogenic causation. Before the emergence of psychoanalytic theory in the 1920s and '30s, however, it was quite clearly considered to be a (central) neurological disorder. This view has recently been forcefully restated in the neurological literature.

Age of onset is rather younger than with other focal dystonias, with the maximum between 20 and 50 years. A marked male preponderance used to be described, probably reflecting the predominance of males in the clerical work force (Lees, 1985). More recent studies report an equal sex distribution (Sheehy & Marsden, 1982). The disorder does not appear to be more frequent in left-handed people (Lees, 1985).

A great deal has been made of the psychogenesis of writers' cramp (Pai, 1947; Crisp & Moldofsky, 1965). While emotional distress may be relevant as a precipitating or exacerbating factor, Sheehy & Marsden (1982) have noted how most patients are exceedingly resilient in dealing with their disability and cannot be considered psychologically exceptional. Furthermore, in their study of 29 cases, these authors used the Present State Examination to study mental state and concluded that "psychiatric factors are either accidental [i.e. no different in type or frequency from the general population] or secondary".

Initial complaints are often vague – usually a recognition of a change in handwriting. Speed of writing may slow or quality of script deteriorate, or a difficulty in moving pen across paper may have been noted. Patients may be aware that they now grip the pen too tightly and push it clumsily through the paper or that they can no longer keep to straight lines. The hand may ache readily. It is important to note that in Sheehy & Marsden's (1982) series only 25% complained of cramps *per se*. Tremor is present in up to 50% of cases, however. (Recently focal task-specific tremor states have also been described both in isolation and in association with task-specific dystonias (Rosenbaum & Jankovic, 1988).) Such complaints are more striking when the patient has to write under pressure, such as during examinations or to meet a deadline (Liversedge, 1961). Writing even single words may become impossible.

Signs are usually striking, with clumsiness in grasping the writing implement, a tight grasp and exaggeration of the normal, semiflexed posture of the digits and hyperextension of the distal interphalangeal joint of the index finger. With writing, typical dystonic postures develop in fingers, wrists and arm, so that the fingers extend, the wrist lifts from its resting surface, the elbow elevates and the forearm pronates (Marsden, 1986). Reduction of arm swing on walking may be noted on the affected side, but other objective signs are usually lacking.

'Tricks' to help writing include exclusion of the index finger in holding the pen, switching to a thicker implement such as a felt-tip pen, using the unaffected hand to 'guide' the pen, or writing standing up. Half of Sheehy & Marsden's patients learned to write with the other hand, but a third of these developed dystonic problems on the contralateral side also. Even when writing on paper is impossible, subjects may experience no difficulty writing 'free-hand' in air or on a blackboard (Liversedge, 1961; Lees, 1985).

The problem is intermittent from the outset in only about 25% of cases. Some begin and remain as *simple* writers' cramp (i.e. the difficulty affects writing only); others show features of *dystonic* writers' cramp from the start (i.e. spasms affecting other dexterous tasks). About a third of 'simple' cases progress to the 'dystonic' form of the disorder, sometimes after many years. Although spontaneous partial remissions have been described, no patient in Sheehy & Marsden's series experienced any prolonged remission. This is therefore a disabling disorder with potentially serious employment consequences.

A great variety of other so-called occupational dystonias or 'craft palsies' have been described (Hunter (1978) for example listed 55). Some of these, because of changing work practices, are now rarely, if ever, seen – for example dystonias in telegraphists, formerly a major problem (Ferguson, 1971), and in money-counters. Others still seen include typists' dystonia, foot dystonias in ballet dancers, dystonias in musicians, especially pianists (Hochberg *et al*, 1983), and loss of dexterity in professional sportsmen whose sport involves the use of an implement, such as golfers, and darts and snooker players.

All these conditions share in common the breakdown of a particular motor skill essential for the normal exercise of the individual's occupation (Lees, 1985). In such cases, traumatic and orthopaedic causes of dysfunction must be carefully excluded.

Laryngopharyngeal dystonia

Dystonia involving laryngopharyngeal muscles is rare but can cause dysphonia and dysphagia (Marsden, 1976*a*, 1986; Lees, 1985).

The major type of laryngeal involvement is an action dystonia resulting in abductor spasm with attempted phonation (Fahn,1984). Speech is strained, hoarse, and staccato ('spastic' or 'spasmodic' dysphonia) and there may be a sense of tightness in the throat. A relationship with Meige's syndrome has been suggested (Marsden & Sheehy, 1982). Less common is the soft, low-volume, 'breathless' speech of laryngeal abductor spasm. Although uncommon, the author has seen laryngeal abductor spasm in the context of neuroleptic-treated chronic schizophrenia, where the 'silly' speech was attributed to mental state.

What may be more common in neuroleptic-treated patients is a tensing of oropharyngeal muscles on talking, resulting in thick speech with slurred, indistinct diction, but little or no disturbance of pitch or inflection. Parkinsonism may contribute to this disorder and it is often difficult to exclude the intruding effects of other established oral dyskinesias, but it is likely that pharyngeal dystonia can present thus more often than is acknowledged.

Segmental dystonias

Many combinations of contiguous involvement can be encountered. Only Meige's syndrome will be mentioned here.

Meige's syndrome

For such a comparatively rare disorder, the syndrome described by Meige in 1910 as 'spasme faciale mediane' (Meige, 1910; Tolosa & Klawans, 1979) is known by a confusing array of names. Perhaps the most satisfactory is the descriptive one: 'blepharospasm - oromandibular dystonia syndrome', although the eponym (or one of the several eponyms) is now often applied when only one of the constituent features is present (blepharospasm or oromandibular dystonia). In contrast to the virtual neglect of Meige's paper in the past, the last 15 years have seen a burgeoning of interest in the condition (Paulson, 1972; Marsden, 1976*b*; Tolosa, 1981; Stahl *et al*, 1982; Jankovic & Ford, 1983; Tolosa & Marti, 1988). With increasing recognition it has been suggested that this syndrome may be as common as spasmodic torticollis (Lees, 1985).

Peak onset is in the sixth decade and there is a female excess (Marsden, 1976*b*; Jankovic, 1988*b*). The blepharospasm component is no different clinically from that described above. In Marsden's series of 39 cases of what he referred to as Brueghel's syndrome, a third had blepharospasm alone. Thus this syndrome increasingly appears to be incorporating cases of so-called 'essential' blepharospasm, though as was noted, when examined carefully and repeatedly, the great majority of patients with blepharospasm will eventually show signs of other craniocervical dystonia.

The oromandibular component comprises lip pursing, 'bridling' retraction of the corners of the mouth, forced mouth opening either in the midline or with lateral jaw displacement, tongue protrusion, and spasms of the floor of the mouth. The platysma may also be involved. Spasms are of varying duration and can contort the whole face into a disfiguring expression of revulsion and can be sufficiently violent to dislocate the jaw (Marsden, 1986). Extension to other areas may occur, resulting, for example, in grunts and snorts similar to those found in Gilles de la Tourette's syndrome and arising from involvement of respiratory musculature. Involuntary levator inhibition, torticollis, spasmodic dysphonia from laryngeal adductor spasm and postural tremor are all regularly encountered (Marsden & Sheehy, 1982; Tolosa & Marti, 1988; Jankovic, 1988*b*). The same 'triggers' noted for blepharospasm may precipitate facial contortions which in turn may be alleviated by 'sensory tricks'.

Blepharospasm–oromandibular dystonia syndrome usually has no identifiable cause (primary Meige's syndrome). Increasingly however these features are being noted as secondary phenomena, especially to drug ingestion. The term 'secondary Meige's syndrome' has been applied in this context but the eponym, if used at all, is best reserved for the idiopathic variety (Jankovic, 1988*b*; Burke & Kang, 1988). Neuroleptic drugs are the most commonly implicated pharmacological agents (Weiner *et al*, 1981; Burke *et al*, 1982) but others include L-dopa in the treatment of Parkinson's disease (Weiner & Nausieda, 1982), antihistamine decongestants (Powers, 1982), and amphetamines (Jankovic, 1981). As noted above however (cf. Classification) a range of neurological disorders must also be considered.

Generalised dystonia

Generalised dystonia may be symptomatic or idiopathic. Many, although not all, cases of idiopathic generalised dystonia are familial, with an autosomal dominant pattern of transmission demonstrated in many families (Zeman & Dyken, 1968). Furthermore, in Ashkenazi Jews, who appear to have a higher prevalence of this disorder than other ethnic groups (Fahn *et al*, 1987), an autosomal recessive pattern has been postulated, although this remains somewhat controversial. An X-linked pattern has also been found but only in the Philippines (Lee *et al*, 1976). The dystonia gene may be more widely distributed than has thus far been appreciated, especially if all dystonias are considered as a unified nosological entity.

Idiopathic generalised dystonia is an uncommon disorder, usually beginning in childhood or adolescence and progressing to severe disability. It is unlikely to be encountered in a primarily psychiatric setting.

Drug-related (symptomatic) dystonias

'Tardive' dystonia

The term 'dystonia tarda' (i.e. dystonia associated with prolonged exposure to antidopaminergic drugs) was first used by Keegan & Rajput (1973). These authors were drawing attention to persistent dystonic postures, which, although noted in some previous studies of drug-related involuntary movement disorders, had largely been overlooked. It is only in recent years that 'tardive dystonia' has been given much consideration (Burke *et al*, 1982). This is in striking contrast to the other varieties of involuntary movement associated with long-term neuroleptic exposure.

The general impression is that this disorder is rare (Gerlach, 1979; J. Kane quoted in Burke & Kang, 1988). In one of our studies, using the 43-item Rockland or Simpson Dyskinesia Rating Scale, which allows for rating of at least some dystonic forms, we found that this type of abnormality was recorded infrequently in a sample of 411 chronic schizophrenics (Owens, 1985). Ankle rotation was the most frequently noted abnormality (5.4%), while retrocollis, torticollis, and axial dystonia were noted only in 0.7%, 1.5%, and 2.7% respectively. Since 47 patients in this sample had no history of neuroleptic exposure, some of the abnormality included in these figures may represent spontaneous idiopathic disorder. By contrast, the drug-related component may have been under-represented by virtue of the group being relatively elderly (see below). Nonetheless, figures of this order are in broad general agreement with those of Yassa *et al* (1986), who found an overall prevalence of 2% in a similar population using the same rating technique. Such figures, and assumptions about the infrequency of the disorder, must however be viewed critically. Most established dyskinesia rating scales are designed to rate overwhelmingly hyperkinetic-type disorder, not static postural distortions. If the history of other forms of movement disorder in psychotic patients is to be repeated, it is very likely that increasing awareness and skill in recognition, especially of the subtler dystonic manifestations, will lead to greater prevalence rates being reported.

Tardive dystonia does not differ from what has already been described, and the same terminology is appropriate. Axial dystonia imparting a fixed rotational lateral tilt to the trunk, when unilateral, has been referred to as the 'Pisa syndrome' (Ekbom *et al*, 1972; Gerlach, 1979) because of the patient's characteristic truncal lean. The author has also seen bilateral involvement with resultant hyperextension of the spine, accentuated lumbar lordosis, compensatory increase in length of step, and a marked subjective feeling of instability, especially where gait could not be altered, for example when descending stairs. The 'Pisa syndrome ' is not however a separate entity, and the name is best dropped.

The majority of tardive dystonias (perhaps up to two-thirds of cases) begin in the muscles of the face and neck, although most patients subsequently develop disorders elsewhere (Burke & Kang, 1988). Isolated focal craniocervical dystonias appear to be uncommon. At point of maximum severity a segmental pattern of distribution is usual (Kang *et al*, 1986). Retrocollis was a particularly characteristic part of the clinical picture in Kang *et al*'s patients, being present in approximately 40% of cases.

Onset tends to be gradual after months or more commonly years of treatment with dopamine antagonists, although atypical onset after only a few weeks or even days of exposure has been described (Burke *et al*, 1982). As in other contexts, 'tricks' may help and activity exacerbate the disorder. Progression does not appear to be inexorable in that most patients seem to reach a plateau of severity (Burke & Kang, 1988), although outcome in terms of severity, distribution, functional disability or reversibility cannot be predicted. Functional disability is likely to be greater with dystonic than with choreiform disorder.

An important factor in the clinical expression of tardive dystonia is age. Prevalence appears to increase linearly with age (Kang *et al*, 1986). However, it also seems that patients with tardive dystonia alone are younger than those with other manifestations of neuroleptic-related hyperkinesis but no dystonia (Gimenez-Roldan *et al*, 1985; Yassa *et al*, 1986). Hence the cumulative distribution curve for tardive dystonia with age appears 'shifted' to the left compared with that for 'classic' tardive dyskinesia. Furthermore, distribution is to some extent related to age of onset. As with idiopathic dystonia, the younger the subject at onset the greater the likelihood of generalisation (Burke *et al*, 1982; Kang *et al*, 1986). In common with other neuroleptic-related movement disorders, however, there is no proven association with type of neuroleptic, dosage, or duration of exposure. Also there is no known difference in prevalence between the sexes.

Other manifestations of neuroleptic-related movement disorder may be present, such as choreiform, tic-like and myoclonic dyskinesias, as well as tremor. In about half of Kang *et al*'s sample, however, dystonia presented in the absence of a history of orolinguomasticatory dyskinesia.

Neuroleptic-related dystonia remains a relatively 'new' entity. Most of the studies have so far come from neurological centres which are likely to experience differential referral predicated largely on the referring agent's curiosity or the patients' functional disability. Studies with their emphasis on 'bizarre' or disabling kinetic disorder are unlikely to provide a balanced picture of the extent of the problem. It is very probable that a large 'pool' of static dystonic postural abnormalities is lurking within the long-term psychotic population, and psychiatrists must now add this to the differential list of motor disorders, long known but oft forgotten, to be found in such patients (Marsden *et al*, 1975; Owens *et al*, 1982; Rogers & Hymas, 1988).

Other drug-related dystonias

Dystonia as both an early and late manifestation of untreated Parkinson's disease has been known for about a century (Gowers, 1888; Purves Stewart, 1898). It is also however a well described complication of L-dopa therapy of this condition, its emergence apparently relating to the periodicity of the clinical effects of the drug (Poewe *et al*, 1988).

'Off-period' dystonia is commonest, affecting up to a third of patients treated with L-dopa for longer than three years (Nausieda *et al*, 1980; Curtis *et al*, 1984). It occurs when the drug's effectiveness is wearing off or at its lowest. It is especially likely to occur in the morning before first dose and has a predilection for the legs (Poewe *et al*, 1988). 'Biphasic' dystonia occurs at onset and waning of drug efficacy and tends to have a unilateral distribution, while 'peak-dose' occurs at the height of anti-Parkinsonian activity and most often involves craniocervical areas (Poewe *et al*, 1988). These phenomena do not appear to relate to the duration of treatment but seem to affect those with early-onset Parkinsonism (under 50 years). Management that does not involve some compromise of anti-Parkinsonian efficacy is difficult.

Dystonia occurring with diphenylhydantoin treatment is confined to patients with plasma levels in the toxic range, occurs with other dyskinesias, and is readily reversible with dose reduction (Chadwick *et al*, 1976). Rarely, dystonia has been attributed to carbamazepine administration, although only as a transient or readily reversible phenomenon in severely brain-damaged children (Crosley & Swender, 1979) or young adults (Jacome, 1979). Movements of a more jerky choreoathetoid type have been noted with dextroamphetamine administration in hyperactive children (Mattson & Calverley, 1968).

Psychogenic dystonia

As will be evident from the above, neurologists have in recent years reasserted their traditional suzerainty over the dystonias, largely by unifying a diverse range of clinical disorders into a classificatory system that acknowledges a spectrum of illness. This reassertion is forceful, but not dogmatic, and leaves open the question of whether dystonia can be psychogenic.

Cases of factitious or willed postural disturbance, admitted by the patient, producing appearances similar to those of dystonia, have been described (Lesser & Fahn, 1978; Batshaw *et al*, 1985). If strictly defined as an *involuntary* disorder, however, such cases cannot be considered as authentic dystonia. Rather, they are consciously simulated. The term psychogenic *dystonia* must be reserved for examples of conversion hysteria.

Conversion hysteria causes many psychiatrists great angst. As a concept, it is one of the cornerstones of psychiatry. As a diagnosis it turns out to be wrong with disconcerting regularity. The balance of opinion is that psychogenic dystonia as a manifestation of conversion is very rare. In the largest published series of patients with dystonia, comprising 932 cases from the Dystonia Clinical Research Center in New York, 24 (or 2.6%) were diagnosed as psychogenic (Fahn *et al*, 1987). It is unclear however if this included simulated dystonia. Hence the true figure for 'authentic' conversion reactions may be even lower.

Despite this, surveys suggest that 40% of patients with dystonia are given a diagnosis of conversion hysteria at some point (Fahn *et al*, 1983), although not always by psychiatrists. This need not necessarily be interpreted as meaning that psychiatrists 'get it wrong' 40% of the time, but may be seen as reflecting a different orientation to a complex clinical phenomenon in which psychogenic factors are clearly important in expression, control, and adaptation, if less so in actual cause. The alternative explanation is, of course, that in such cases psychiatrists 'get it wrong' 40% of the time!

The diagnosis of conversion is as much related to characteristics of the diagnostician as it is to the features being diagnosed, and the literature offers little guidance to help the analytically overcommitted offset their backgrounds. Fluctuating neurological signs, such as shifting or non-dermatomal sensory disturbance, inappropriate weakness ('giving way'), pain on light touch or possibly evidence of factitious abnormality elsewhere have been cited (Fahn *et al*, 1983) but are themselves in large part subjective in their evaluation. Perhaps the best parameters are the lack of a normal progression of symptoms with, for example, no phase of 'action' dystonia, and a presentation dominated by static postural disturbance with a relative absence of typical hyperkinesis.

Even here, however, pitfalls arise. The author has seen one patient with sudden evolution of severe torticollis over a few days following a break-in and attempted rape at gun-point in her New York apartment. She felt 'cured' by behavioural and relaxation techniques and was psychologically exceedingly well adjusted after treatment, but clearly showed continuing minimal head rotation with unequivocal scoliosis when examined four months later. She was quite unaware of this. These postural abnormalities were also evident in family photographs two years afterwards.

In the author's opinion, psychiatrists should be highly circumspect in attributing any dystonic abnormality to a purely psychogenic causation.

Diagnosis

Diagnosis of dystonia is based almost entirely on the clinical appearance. Family history is usually unhelpful, except in the rare genetic forms, which are unlikely to present psychiatrically. Nonetheless, psychiatrists must bear in mind the range of disorders that can present with dystonia even if precise diagnosis and management of these is best undertaken in a neurological centre. The most important factor for psychiatrists to evaluate is drug history, especially of antipsychotics.

It is essential that psychiatrists remain aware of the unique characteristics of this type of involuntary movement in order to guard against overzealous and often misguided application of analytical theory.

Pathology/pathophysiology

No consistent anatomical lesion has been identified in dystonia. Studying cases of symptomatic hemidystonia, Marsden *et al* (1985) suggest that the underlying problem emanates from a disruption of input from basal ganglia (thalamus, caudate and putamen) to pre-motor and ultimately motor cortex: dystonia is therefore seen as the result of release of pre-motor cortex from thalamic control.

At the neurochemical level a number of theories have been proposed, usually relating to hypothesised disruption of central dopaminergic/cholinergic balance, but no consistent data have as yet emerged to allow construction of a coherent hypothesis. There is evidence that acute dystonic reactions may result from a complex interplay of factors involving presynaptic dopaminergic mechanisms (autoreceptors) as well as post-synaptic receptors (Marsden & Jenner, 1980).

Treatment

The treatment of dystonia is unsatisfactory. Evaluation of any treatment programme must take account of the bizarre characteristics of the disorder, the fluctuating intensity of symptoms, the occasional tendency to spontaneous remission and the non-specific benefits of medication and authoritative professional involvement. Evaluation must also involve the use of standardised techniques for staging the disorder, reliably applied. For these reasons such an uncommon disorder is best investigated and managed in a specialist centre where adequate numbers of patients can be studied against suitable controls. Further single case studies will extend our knowledge but little.

Treatment of idiopathic dystonias

Drug treatments

Representatives of virtually every class of centrally acting drug have been tried. Sheehy & Marsden (1982), for example, note 23 preparations tried in their cases of writers' cramp alone (for a comprehensive list of preparations used in different forms of dystonia see Marsden (1986)). Response to medication is unpredictable, that which benefits one case exacerbating or being ineffective in another. This pharmacological variability may have implications for future classificatory systems.

The most encouraging therapeutic development in recent years has been the efficacy of high-dose anticholinergics in both open (Fahn, 1983; Marsden *et al*, 1984) and double-blind (Burke *et al*, 1986) trials. Benzhexol (trihexyphenidyl) introduced in low/average doses of 2–6 mg daily has been most extensively tested. The dose is increased by the order of 2 mg per week to tolerance or significant improvement. Using this slow incremental regime very high doses have been tolerated (Marsden *et al* (1984) report a maximum of 130 mg daily), though with great individual variability. Average doses range from 20 to 40 mg per day. In general, 50% of children and 40% of adults with idiopathic dystonia obtain at least moderate benefit from high-dose anticholinergics (Fahn & Marsden, 1987). The better response in children may reflect the lower prevalence of central side-effects in them than in adults, and hence their greater tolerance to such regimes (Fahn, 1983; Fahn & Marsden, 1987). The theoretical risk of problems with memory and learning, especially in children, has not yet been reported. It is interesting to note the relative infrequency of anticholinergic psychoses with this slow method of dose increment. The only predictor of response identified so far is early initiation of treatment, with a better response claimed for those started on treatment within five years of onset of dystonia (Fahn & Marsden, 1987).

How anticholinergics work in this situation, why high dosage has to be employed, and why it can take months for improvement to become evident are all unknown, but this at least offers some therapeutic prospects for sufferers of an otherwise disabling and distressing disorder.

Psychological/psychiatric treatment

One of the more important therapeutic influences on dystonia is the physician–patient interaction. Patients are often frightened and confused by this uncontrollable disability which behaves so strangely, and may themselves believe, or may have been told by doctors, that it represents a psychological disorder. Initial medical reaction is important, and should referral be to a psychiatrist it is essential that, before plunging into the recesses of a supposedly disordered subconscious, the almost exclusively medical nature of the problem is openly acknowledged. Patients are invariably more stoical in dealing with the disabilities of an illness whose mysterious origins and empirical treatments are admitted than they are at coping with the tyranny of an imposed psychopathology.

This must not be taken as meaning that psychological/psychiatric interventions have nothing to offer. This is certainly not the case, and in this regard neurologists, by their own admission, may have swung the pendulum too far the other way, as hinted by Fahn *et al* (1987).

A range of psychological treatments have had, and still have, their advocates. The problem is that most reports of efficacy in this field concern single case studies or small patient groups. No controlled trials or long-term follow-up studies of adequate samples have been undertaken. Nonetheless, substantial improvements have been reported with a range of mainly behavioural techniques (Lees, 1985; Faulstich *et al*, 1985; Fahn & Marsden, 1987). The details of these are beyond the scope of this article, but include relaxation (in its many forms), negative practice, and biofeedback. It is likely that the beneficial effects of such programmes lie less in the theoretical constructs behind them than in their non-specific influences on mental state, muscle control, composure, and overall ability to cope. Whatever their mode of action and efficacy – and usually in the context of an acknowledged medical disorder – they are certainly worth trying in skilled hands, when other treatments are of such limited value.

It is perhaps axiomatic to state that any coexistent psychiatric illness should be conventionally managed. Supportive psychotherapy is also a helpful adjunct (Fahn & Marsden, 1987) by providing education, advice, and understanding where all of these may be lacking elsewhere. Prolonged or interpretative psychotherapies have no place in management in the absence of clear psychogenesis. Mostly these are unhelpful, although in some cases where the medical nature of the disorder has not been recognised and treatment has consisted of a stubborn insistence on the development of dynamic insight, they have been clearly detrimental (Eldridge *et al*, 1969; Fahn & Marsden, 1987).

Surgical treatment

Detailed consideration of this highly specialised area is beyond the scope of this article. Surgical procedures range from myectomy, through peripheral nerve and spinal root section, to thalamotomy. Results are variable (Fahn & Marsden, 1987).

An interesting recent innovation, especially in the treatment of blepharospasm, is local (supraorbital) injection of botulinum A toxin (Jankovic, 1988*c*). This is the most potent biological toxin known and was originally introduced in 1980 to treat strabismus. It acts by rapidly and irreversibly binding to presynaptic cholinergic membranes, from where it is internalised, thereby disrupting calcium influx (Jankovic, 1988*c*). Good results have been achieved with few local side-effects, although treatment may have to be repeated.

Treatment of 'tardive' dystonia

Tardive dystonia presents a singular management problem. This relates to the greater functional disability than that associated with other drug-related dyskinesias, the likelihood of sufferers being younger, potential irreversibility and the requirements for long-term medication to maintain psychiatric well-being.

The crucial first-step is a re-evaluation of the need for on-going neuroleptic treatment. A feature of American work is the relatively high proportion of patients with tardive dystonia who had been treated with neuroleptic drugs for non-specific and non-psychotic psychiatric states (Burke *et al*, 1982): 22% of Burke & Kang's (1988) patients had received these drugs for anxiety, multiple somatic complaints, nausea, etc. The situation would (hopefully) be rather different in the UK. Neuroleptics are indicated for non-psychotic states in extreme circumstances only and should be discontinued if dystonia ensues.

Most patients, however, will be schizophrenic. Stable, functioning patients with a history of only one episode may not benefit from prophylaxis (Crow *et al*, 1986) and may therefore survive discontinuation of antipsychotics. Since it is not possible to identify relapsers in this group (MacMillan *et al*, 1986) such patients who develop dystonia are entitled to a trial drug-free period to assess both the response of their neurological signs and their relapse potential. It is important to note that Burke & Kang (1988) have reported that the only patients in their series (with mixed diagnoses) whose dystonia resolved completely came from the ranks of those who came off, and could remain off, antidopaminergic medication.

For those with an established pattern of relapse on neuroleptic withdrawal discontinuation is hardly an option, even with such a problem as dystonia. It is then worthwhile to re-evaluate dosage, and reduce to the minimum to prevent relapse. As noted, there is no evidence linking dystonia to antipsychotic drug dosage, but such a step would seem clinically reasonable if not scientifically logical. It is certainly good practice. Because of the delay of any possible beneficial effects from such a move 'filtering through' with depot preparations, the author prefers if possible to switch to an oral preparation in this situation, although this is a personal rather than a proven manoeuvre.

If dystonia persists, specific interventions may be necessary. High-dose anticholinergic as described above may produce improvement in up to half of cases (Kang *et al*, 1986; Burke & Kang, 1988). Exacerbation of coexisting 'classic' (choreoathetoid) tardive dyskinesia with anticholinergic treatment seems more a theoretical than a practical risk, as confirmed in Kang *et al*'s (1986) series, where only one of ten patients showed exacerbation of complex oral dyskinesias.

Alternatively addition of a pre-synaptic catecholamine-depleting agent such as reserpine or tetrabenazine may be helpful (Burke & Kang, 1988) but side-effects such as depression or disabling Parkinsonism may limit their usefulness. Nonetheless, Kang *et al* (1986) found these to be the most useful agents in their dystonic group, although two-thirds of this sample did not have a clear psychotic diagnosis. Depleting agents may be a less practical option in a purely schizophrenic group.

Finally, in 'classic' tardive dyskinesia, increasing the dose of antidopaminergic medication may suppress the movement disorder (American Psychiatric Association, 1979; Jeste & Wyatt, 1982), and where all else fails, this may be considered with drug-related dystonia. No published work to date has addressed this. The theoretical risk of such an approach is that of subsequent 'breakthrough' of abnormal movements at a later date, possibly with greater severity. This is based on assumptions drawn from the post-synaptic dopamine receptor supersensitivity theory of the pathophysiology of tardive dyskinesia. This is of course only a theory which accommodates some, but not all, of the clinical observations concerning drug-related movements, and it is one that has been challenged (Jeste & Wyatt, 1981). To date there is no clinical evidence that this 'breakthrough' actually occurs. Nonetheless, such a strategy is probably best reserved for cases of severe disability or generalised dystonia when all else has failed. When this degree of disorder is associated with evidence of muscle damage (with elevated levels of creatinine phosphokinase) one is dealing with a potentially life-threatening situation (Lazarus & Toglia, 1985; Burke & Kang, 1988) and extreme measures are justifiable.

Conclusion

Dystonia has recently undergone a substantial revision in the neurological literature. The purpose of this article has been to highlight this to a psychiatric audience who, by tradition and inclination, may tend to see this disorder from a different perspective. Knowledge remains scant and it would be wrong to assume that neurologists have a monopoly of insights into these perplexing states. Nonetheless, as Lees (1985) has pointed out, although the evidence linking the disparate presentations of dystonia to each other and ultimately to disorder of the basal ganglia is circumstantial, it is nonetheless persuasive. Psychiatrists would do well to be familiar with it.

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Lecture

Alcohol and the Brain*

W. A. LISHMAN

Some of the major effects of alcohol, and alcoholism, on the brain are reviewed, with reappraisal of evidence drawn from brain imaging, neuropathology, clinical psychology, and laboratory experimental work. A hypothesis is developed which may help to account for the wide variability encountered in individual susceptibility to alcoholic brain damage and its varied manifestations. Therapeutic implications are considered.

The first Benno Pollak lecture dealt with public health and social policy issues in the field of alcoholism (Kendell, 1987), the second with the long-term course of the alcoholic career (Edwards, 1989). This, the third, addresses what we may broadly term 'physiological' studies in the realm of alcoholism by considering some of its major effects on the brain. In part it reviews work over the past decade in the Section of Neuropsychiatry of the Institute of Psychiatry. The topic illustrates certain broad themes and principles in medicine and in scientific progress generally – principles with wide applicability but perhaps particularly well displayed in the present context.

The oversimplification of medical conceptions

The first theme concerns the tendency always present for medical conceptions to be most attractive when they are relatively simple, and neatly sewn together. We need to proceed in this way in order to make sense of the complexities underlying pathological states, and nowhere more so than in disorders of the brain. Simple formulas are well remembered and readily perpetuated. Medical teaching, in consequence, can run into the danger of becoming to some extent 'fossilised'.

Alcoholic brain damage was, until fairly recent times, viewed in rather straightforward terms, which may be summarized as follows.

(a) The classic form of brain damage in alcoholics has long been regarded as the Wernicke–Korsakoff syndrome, founded on thiamine deficiency. The alcoholic neglects his diet and runs seriously short of this essential vitamin. What thiamine he does ingest is poorly absorbed, and is soon utilised for the metabolism of alcohol. Both Wernicke's encephalopathy and Korsakoff's syndrome are viewed as the two components, the acute and the chronic, of the same pathological process, affecting the same crucial regions in the hypothalamus, diencephalon, and upper brain-stem.

(b) Wernicke's encephalopathy and Korsakoff's syndrome each have their distinctive manifestations, allowing them, it is said, to be diagnosed with fair reliability during life. Wernicke's encephalopathy consists of the striking conjunction of acute confusion, impairment of consciousness, ophthalmoplegias, and ataxia; Korsakoff's syndrome of chronic mental impairment in which memory deficits overshadow other aspects of cognitive failure.

(c) The Wernicke–Korsakoff syndrome was held to account for the great majority of alcoholics who suffered long-term cognitive impairment, those who escaped this development being regarded as essentially 'intact'. A small and uncertain proportion might suffer alcoholic 'deterioration' or 'dementia', but these were nebulous conceptions. They presumably depended on widespread cerebral dysfunction of an uncertain nature. Neuropathologists were in fact remarkably divided on the issue of diffuse cerebral change in alcoholic subjects, some finding evidence of brain atrophy and cell loss while others disputed such claims. Attention to the alcoholic brain at autopsy remained firmly focused on the basal brain regions.

Reasons have arisen, however, to doubt whether the above is sufficient to explain the alcoholic's problems. Thus Wernicke's encephalopathy is the usual, but not the inevitable forerunner of the chronic Korsakoff state. Moreover, chronic Korsakoff's syndrome does not respond very well, indeed often not at all, to intensive thiamine replacement. Evidence continued to accumulate, from autopsy and air encephalographic studies, to suggest that the alcoholic's brain could be diffusely damaged (Ron, 1977), even when there had been no hint of a Korsakovian development. Courville (1955) suggested that alcoholism was the commonest cause of diffuse cerebral atrophy at autopsy in men under the age of 50. Clinical psychologists began to stress that a high proportion of alcoholics showed very definite and lasting cognitive impairments, even after considerable periods of abstinence. Sometimes the picture resembled dementia.

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*The Third Benno Pollak Lecture, delivered at the Institute of Psychiatry, 13 June 1989.

The possibility of a dementia attributable in some degree of alcoholism certainly persisted in the minds of many clinicians. In repeated series of patients presenting to clinics and hospitals, alcoholism has been suspected of playing a part just as commonly as vascular disease (Lishman, 1981, 1986).

It seemed difficult, however, to carry the matter further, particularly in the absence of a firm consensus from brain post-mortem findings. Victor & Adams (1985) concluded that "the concept of a primary alcoholic dementia must remain ambiguous until such time as its morphologic basis is established."

The promise of new technologies

In the face of such uncertainties the advent of new technologies brings immediate promise. Computerised tomography (CT) was therefore soon applied to the problem of the alcoholic brain: it is non-invasive and could be carried out on random cohorts of alcoholic patients, unlike the earlier neuroradiological techniques. It could, moreover, be viewed in relation to the current clinical picture, and be repeated if necessary at later stages of the disorder. Studies were quickly underway in Toronto (Carlen *et al*, 1978; Carlen & Wilkinson, 1980), Stockholm (Bergman *et al*, 1980), Western Australia (Cala *et al*, 1980), and Camberwell (Ron *et al*, 1979, 1980, 1982). Our own investigations commenced as soon as the first scanner was installed at the Maudsley Hospital some ten years ago. Dr Maria Ron and Dr William Acker collaborated closely with Dr Kenneth Shaw at Bexley Hospital.

The older uncertainties were quickly clarified. Brain shrinkage was detectable in a high proportion of alcoholic patients, even those who seemed at first sight to have escaped cerebral involvement. Our first 100 'intact' patients at Bexley Hospital showed decisive ventricular enlargement, and widening of the fissures and sulci over the hemispheres (Ron *et al*, 1980, 1982). Approximately one-half to two-thirds showed changes which could be regarded as definitely beyond the normal range. By the same token, it should be noted, the remainder appeared to be quite normal.

The natural history of such shrinkage was followed in over half the sample by re-scanning six months to three years later (Ron *et al*, 1982; Ron, 1983). In the majority appearances were unchanged, although some showed worsening, and an important group showed regression of the shrinkage. The factor clearly related to possibilities of improvement was the degree of abstinence achieved during the interim period. With prolonged abstinence the changes were reversible at least in part. In this we confirmed the findings of others (Carlen *et al*, 1978; Carlen & Wilkinson, 1980).

The situation was further checked in a group of patients drawn from Alcoholics Anonymous (AA)

TABLE I

Brain CT measurements in alcoholic patients and controls

	Males			*Females*		
	Private patients	*Bexley*[1] *patients*	*Controls*[1]	*Private patients*	*Bexley patients*	*Controls*
Number	16	100	50	15	22	26
Age: years	45.1	43.5	41.5	41.4	42.3	40.5
Mean ventricular size	9.8	9.6	6.2	7.1	7.9	5.4
(ventricular : brain ratio) (s.d.)	(4.8)	(3.0)	(2.0)	(3.1)	(3.8)	(2.2)
Sulcal width						
grades 0, 1	14	56	48	13	21	26
grades 2, 3	2	43	2	2	1	0
Sylvian fissures						
grades 0, 1	15	86	50	15	21	26
grade 2	1	14	0	0	1	0
Interhemispheric fissure:						
grade 0	13	71	46	12	14	26
grade 1	3	29	4	3	8	0

1. Data from Ron *et al* (1982).
Other data from Jacobson & Acker (unpublished).

(Jacobson, 1986; Lishman *et al*, 1987). Despite severe earlier drinking histories their scans were closer to those of normal controls than to those of current alcoholics; some degree of residual ventricular dilatation was suspected, however, being quite marked in certain cases.

Alcohol neurotoxicity thus emerges as a likely factor in causing, or at least contributing to, the cerebral shrinkage. However, other factors deriving from the hazardous life-style of the alcoholic (anoxia, head trauma, malnutrition) could also play a part. As a potential means of approaching this issue in an economical manner Robin Jacobson and Clare Acker examined a small group of patients from Galsworthy House, a private treatment facility for patients with drinking problems. Although drawn largely from professional and managerial occupations, such patients showed ventricular dilatation quite as severe as in the Bexley samples (Table I).

There is now a considerable literature on experimental approaches to alcohol neurotoxicity, with evidence that prolonged exposure does indeed damage neural elements within the brain (Walker *et al*, 1980*a*,*b*; West *et al*, 1982). Of particular relevance in the present context is McMullen *et al*'s (1984) demonstration that five months of exposure to alcohol in the rat causes a significant reduction of branching of the dendritic domains in the hippocampus, and a reduction of thickness in the corresponding dendritic strata. However, two months of abstinence allows regrowth of dendritic branching and a return to normal thickness of the strata. This provides a tempting model for what we have observed on the CT scan.

In summary, therefore, CT scanning has shown that brain shrinkage is present in a high proportion of alcoholics, affecting both cortical and subcortical regions. It is already evident in quite young subjects, and is seen in 'intact' patients, not merely those who are 'deteriorated'. It is potentially reversible in some degree with abstinence for several months or years, but even after several years the brain may remain abnormal.

Support from neuropathology

Thus far you may be wondering, as we so often wondered, why the brain shrinkage revealed on the scan had not been more clearly evident to neuropathologists: could the CT findings be artifactual in some way? One felt insecure without firm findings from post-mortem studies. In fact recent neuropathological surveys have confirmed that the findings are real, the reappraisal having been provoked in part by the CT findings.

Here we find an illustration of the virtues of measurement when a subject has been open to dispute. Just as painstaking measurements were applied to the brain of the experimental rat, so they were applied to the human alcoholic by pathologists in Australia and Norway. The results have been informative. In comparison with controls, brain weight is slightly but significantly reduced in the alcoholic at necropsy (Harper & Blumbergs, 1982; Torvik *et al*, 1982), and the pericerebral space over the cortex is considerably enlarged (Harper & Kril, 1985). This emerges whether or not there is coexistent evidence of nutritional brain damage, perhaps again pointing to the role of alcohol neurotoxicity. The amount of white matter in the cerebral hemispheres is reduced, and the ventricles are enlarged by approximately 50%, a figure similar to that derived from CT studies (Harper *et al*, 1985). Finally a start has been made in quantitative assessment of neuronal elements within the brain, and counting techniques have revealed a 22% reduction in the number of neurones in the superior frontal cortex of alcoholics (Harper *et al*, 1987).

Unexplained problems

We can conclude, therefore, that the CT findings are real. But it is also apparent that they do not tell the whole story, because the shrinkage, so very evident, does not correlate well with other important factors. For example, in our samples CT findings showed no relationship to length or severity of drinking history. One might try to explain this on the grounds of unreliable historical data, but a considerable number of patients had entirely normal scans after disastrous, well documented drinking histories. Furthermore we found no striking relationships with psychometric deficits, yet the alcoholics were impaired on many tests. This was a more serious obstacle, in that we thought we might have been detecting the pathological basis of early dementia. Other workers have reported relationships of a sort, but we found that virtually all correlations between CT data and psychometric data disappeared in our patients when due account was taken of age and pre-morbid intelligence (Acker *et al*, 1984).

We therefore asked ourselves whether gross morphological measurements could be missing the essential parameter. Was there any other information that could be extracted from the CT scan? We turned to brain X-ray absorption densities – the Hounsfield numbers themselves from which the scan pictures were reconstructed. These possibly reflected tissue characteristics in some useful fashion. Two approaches were employed: the use of 'region of interest markers' which could be sited over discrete brain areas on a scan displayed at a viewing console (illustrated in Lishman *et al*, 1987), and the elaboration of an extensive computer program for scan analysis which proceeded in a fully automated manner (Baldy *et al*, 1986).

We began to get some interesting findings, but soon stumbled upon one source of artifact after another (Jacobson *et al*, 1985). For example, absorption density proved to be slightly but significantly related to duration of abstinence in the alcoholic patients. Any comparisons would need to take this into account. Moreover, even among the normal controls there was a demonstrable relationship with the date upon which the scan was carried out – for obscure reasons the scanner had yielded slowly mounting values while the various studies were being performed. Finally, the Hounsfield numbers were related to skull size. Here we thought for a while that we had discovered a new principle of over-riding interest, namely that the healthy female brain differed from that of the male in showing higher absorption densities. The artifact, beam hardening dependent on skull cross-sectional area, was discovered in time to prevent an embarrassing publication!

Nevertheless, on taking all such artifacts into account we obtained some findings of interest. Absorption densities measured in the frontal white matter proved to be elevated in our long-abstinent alcoholics at follow-up, also moreover in our AA sample (Lishman *et al*, 1987). An analogous result was found by Gurling *et al* (1986) when comparing monozygotic twins discordant for alcoholism. This raises the possibility that absorption densities may reflect some enduring abnormality in the brains of alcoholics which serves as a marker of their past addiction. Lesch *et al* (1972, 1973), for example, reported altered lipid content of brain white matter in alcoholics at autopsy. If the alcoholic brain is in some manner altered in the long term this could conceivably account, in part at least, for their continuing risk of relapse.

Certain correlations also emerged with psychometric data in the alcoholic subjects (Lishman *et al*, 1987). Verbal fluency, known to be sensitive to frontal lobe lesions, showed a small but significant relationship to density measurements in the frontal white matter. Verbal memory, measured by the Logical Memory Test, showed a significant relationship with density measurements obtained from the medial thalamic regions. Such correlations were small, but the finding in the thalamus also emerged in other studies (Gebhardt *et al*, 1984). It is particularly interesting in that the thalamus is implicated in the Wernicke–Korsakoff lesion, which of course impairs memory function very markedly.

The role of subclinical pathology

So far we have been dealing with subclinical pathology of a sort in the cerebral hemispheres and cortex, and finding it to be much commoner than would have been expected. Harper (1979, 1983) in Australia has directed attention to a similar situation with regard to the parts of the brain harbouring the classic Wernicke–Korsakoff lesion. Pathological changes have proved to be commonly found here at post-mortem when not suspected during life. Thus over the course of nine years, Harper (1983) found that 131 cases of Wernicke's encephalopathy could be diagnosed at autopsy, yet only 26 of these had been suspected during life. The great majority of affected persons were known to be alcoholics. A considerable range was encountered in the acuteness or chronicity of the lesions, but two-thirds of cases showed chronic pathological changes alone. This raises the possibility that thiamine-dependent basal brain damage may exist in many alcoholics long before it is suspected. It could build up surreptitiously throughout, or in stepwise fashion during episodes of subclinical Wernicke's encephalopathy which have been overlooked.

This is an attractive hypothesis in many ways. It could help to explain why memory is so prominently affected in many alcoholics, and why the alcoholic Korsakoff syndrome does not respond well to thiamine – there may often have been an accumulation over years of fixed structural pathology. In effect the florid Wernicke's encephalopathy which precedes many Korsakoff syndromes may reflect merely a small amount of additional pathology, but which has catastrophic consequences. Finally it could explain why a proportion of Korsakoff syndromes appear to evolve gradually.

We have obtained some indirect support from CT studies for the idea that pathological changes may commonly exist in the basal brain regions of alcoholic subjects. Changes in such a location might be expected to lead to widening of the third ventricle and to compromise memory functions. Thus among 39 non-Korsakoff alcoholics, thoroughly detoxified and not suffering from overt brain damage, we have found that performance on a battery of memory tests is significantly related to the width of the third ventricle as measured on the scans (Acker *et al*, 1987). Not dissimilarly Jacobson (1987) divided a sample of non-Korsakoff alcoholics into those with good and poor memory, according to the Logical Memory Test, and compared their scans with those

TABLE II

Brain CT measurements in Korsakoff patients, alcoholics with good and poor memory, and controls (all males)

	Korsakoff patients (n = 25)	*Non-Korsakoff alcoholics*		*Controls (n = 24)*
		Poor memory (n = 12)	*Good memory (n = 12)*	
Age: years	56.1	53.4	51.9	55.3
Logical Memory Test score (delayed)	0.6	5.3**	11.7**	10.3
Mean lateral ventricular size (ventricular : brain ratio) (s.d.)	13.7 (4.9)	12.4 (4.5)	9.1 (4.3)	9.4 (3.4)
Mean third ventricular index (logged distribution) (s.d.)	5.6 (2.2)	4.6* (1.4)	3.6* (0.8)	3.3 (0.9)

* $P<0.05$, two-tailed t-test.
** $P<0.001$, two-tailed t-test.
From Jacobson (1987).

of Korsakoff patients and normal controls (Table II). It can be seen that the ventricles tend to be larger in alcoholics with poor than with good memory, the values in the former approaching the values found in Korsakoff patients and the latter the values found in normal controls. The difference in the third ventricular index exceeded the 0.05 level of statistical significance. Although these are very indirect approaches to the problem, it does seem that impaired memory in the alcoholic subject is related to changes of some sort in the deeper structures of the brain.

It is therefore possible to view the brain of the alcoholic as vulnerable to two relatively distinct forms of pathology - *both well before we have clinical grounds for suspecting them*. The first, the cortical changes, with possible neuronal loss, may owe much to alcohol neurotoxicity. The second, ingravescent pathology in the basal brain regions, will owe most to thiamine deficiency. Korsakoff's syndrome, in effect, may be only the tip of an iceberg, many non-Korsakoff patients being affected, and impaired, by more minor degrees of similar pathology.

Turning to the Korsakoff patient himself there is evidence of both cortical and subcortical damage in substantial degree (Jacobson, 1989; Jacobson & Lishman, 1990). Table III shows that not only is the third ventricle wider than that of matched non-Korsakoff alcoholics, as might have been expected from their diencephalic lesions, but the lateral ventricles are also more dilated and the Sylvian and interhemispheric fissures are wider. Indeed, the scans of many of our Korsakoff patients showed the most severe grades of cortical and subcortical shrinkage we have encountered among alcoholic subjects.

The widening of the interhemispheric fissures between the frontal lobes was particularly marked. This is a reminder that the Korsakoff patient shows not only memory impairment, but also changes of behaviour (apathy, inertia, loss of insight) indicative of frontal dysfunction. The frontal changes may

TABLE III

Brain CT measurements in Korsakoff and non-Korsakoff alcoholics

	Korsakoff patients (n = 25)	*Non-Korsakoff alcoholics (n = 24)*	*Significance*
Age: years	56.1	52.6	NS
Mean third ventricular index (logged distribution) (s.d.)	5.6 (2.2)	4.1 (1.3)	$P<0.001$
Mean lateral ventricular size (ventricular : brain ratio) (s.d.)	13.7 (4.9)	10.7 (4.6)	$P<0.05$
Sulci enlarged	44%	25%	NS
Sylvian fissures enlarged	68%	29%	$P<0.05$
automated pixel count	36.6	18.2	$P<0.05$
Interhemispheric fissures enlarged	80%	33%	$P<0.01$
automated pixel count	45.8	21.6	$P<0.05$

From Jacobson (1989).

contribute to special features of the amnesic syndrome (e.g. failure to release from proactive interference (Moscovitch, 1982), and to other aspects of cognitive impairment. In our sample the Korsakoff patients were inferior to other alcoholics on tests sensitive to frontal lobe lesions, such as verbal fluency and category sorting tests, several aspects of the latter being related to the size of the inter-hemispheric fissure (Jacobson, 1989).

It thus emerges that the Korsakoff patient is likely also to be suffering from this combination of alcohol neurotoxicity and thiamine deficiency. *Both* may need to be manifest in severe degree before the syndrome is encountered in fully fledged form.

Neurochemical considerations

So far we have dealt only with structural brain changes, as revealed by brain imaging or histological examination. We must now consider the important principle that neurochemistry is coupled to brain morphology. Small lesions in the basal brain regions can have profound neurochemical effects on other parts of the brain via pathways which are gradually being defined.

The discovery of the nigrostriatal dopamine pathway set the stage, and pathways involving other neurotransmitter systems were soon delineated. We now know, for example, that the locus coeruleus in the upper brain-stem is the principal source of noradrenergic inputs to the cortex, that the raphe nuclei deal with serotonin, and, very importantly, that the substantia innominata of the basal forebrain contains nuclei which furnish cholinergic inputs into the cortex. The nucleus basalis of Meynert is a principal component of this group.

A great impetus to clinical interest in such systems has come from the recognition of their importance in Alzheimer's dementia. Brain cholinergic depletion of profound degree is the most striking chemical finding in this disorder, and the nucleus basalis of Meynert is known to be depleted of neurons. Vigorous attempts in consequence are afoot to seek means of remedying the biochemical deficits.

Should not such knowledge be applied, just as vigorously, to alcohol-related brain damage? There are indeed good reasons why it should, for the basal brain nuclei stand to be damaged in the alcoholic brain. The thiamine-dependent lesion of the Wernicke–Korsakoff syndrome extends through the upper brain-stem, hypothalamus, and adjacent brain regions. The locus coeruleus can certainly be implicated, and nucleus basalis of Meynert may also suffer damage. How far this last is due to thiamine deficiency, or whether it results from alcohol neurotoxicity, has not been clarified, but cell counting has shown directly that the nucleus basalis of Meynert may be depleted of neurons in Korsakoff's syndrome (Arendt *et al*, 1983). Thiamine deficiency, moreover, can interfere directly with acetylcholine turnover via biochemical pathways.

Kopelman (1985) has carried out a detailed comparison of the memory problems of Korsakoff and Alzheimer patients. They have many deficits in common, with particular difficulty in getting material into long-term storage. This is not, however, in either case, an exact parallel of acetylcholine depletion. Cholinergic blockade does not account, for example, for the deficits in primary memory seen in Alzheimer's disease nor the long retrograde amnesia seen in Korsakoff's syndrome (Kopelman & Corn, 1988). Other factors, perhaps other neurochemical systems, are almost certainly implicated in the memory problems of both disorders. In Korsakoff's syndrome there has been reason to suspect noradrenergic depletion, and improvements in functioning have been reported after treatment with clonidine (McEntee & Mair, 1980; McEntee *et al*, 1984). More recently there has been evidence that fluvoxamine may have a beneficial effect on Korsakoff memory deficits, presumably through its effects on the serotonergic system (Martin *et al*, 1989).

The important point is that Korsakoff's syndrome could yet prove to be amenable, at least in part, to appropriate biochemical manipulation of neuro-transmitter systems. In many ways it would seem a 'better bet' than Alzheimer's disease, in which the pathological process is progressive.

It is worth noting, moreover, that the lesions at the base of the alcoholic's brain, when well developed, may conceivably contribute to a picture which resembles dementia (Lishman, 1966). The evidence for this is slender, but it deserves our attention. Torvik *et al* (1982), in Norway, have shown that some 10% of alcoholics dying in institutions have pathological changes in the Wernicke–Korsakoff region at the base of the brain. In this he confirms Harper's view that such lesions are much commoner than expected. But on examining the clinical histories, where fair documentation existed, he found that many had appeared to be demented. Thus of 20 patients with chronic pathology there, five had shown the selective memory deficits of Korsakoff's syndrome, while 15 had, on the evidence available, suffered "a more or less pronounced global dementia in addition to a severe memory defect". Eight of these patients had been labelled during life as having a pre-senile or senile dementia, but in fact only two showed Alzheimer changes and one the changes of multi-infarct dementia on autopsy. A recent report has, moreover, indicated a depletion of neurons in the nucleus basalis of Meynert in a small group of patients diagnosed as suffering from alcoholic dementia (Akai & Akai, 1989).

Putting such strands of evidence together it would seem that some alcoholics may develop lesions which impair their brain biochemistry to the extent that they develop a global dementia. These again may be important subjects on whom to test the efficacy of pharmacological strategies.

Alcohol toxicity and basal brain changes

So far we have largely assumed that the basal brain changes in the alcoholic are due solely to thiamine deficiency. There is certainly abundant clinical and experimental evidence pointing to its role in producing these lesions in the diencephalon and upper brain stem. But there may be more to the story in the alcoholic subject. The principle illustrated here is the importance of fresh appraisal as newer evidence comes forward.

We have first the intriguing work carried out by Arendt *et al* (1988) on the experimental rat. It was shown that rats fed on alcohol for 28 weeks develop a severe and persistent impairment of maze learning, along with evidence of brain cholinergic depletion. Maze performance was improved, however, by the implantation of choline-rich neuronal tissue into the cortex and hippocampus, the degree of improvement being closely related to the extent of restoration of cholinergic function. Histological study showed that the classic Wernicke-Korsakoff lesion was not present, since the animals were not malnourished, but significant cell loss was detectable in the cholinergic nuclei of the basal forebrain. Thus alcohol itself appears to have been markedly detrimental to this region, with profound neurochemical consequences.

In man the evidence comes from a different approach. Hata *et al* (1987) investigated regional cerebral blood flow by xenon-enhanced CT in alcoholic subjects. Diffuse reductions in flow were seen throughout the cortex and deep grey matter, but the most pronounced effects were in the hypothalamus and basal forebrain, both in Wernicke-Korsakoff *and* non-Korsakoff subjects. With thiamine replacement in the former, and with abstinence in the later, perfusion was partly restored. Although resting on small numbers of subjects, Hata *et al* interpret their results as pointing to a role of alcohol neurotoxicity, even in the absence of thiamine deficiency, in disturbing cerebral perfusion in the basal brain regions.

Conceivably, therefore, it could be a *dual vulnerability* of subcortical nuclear masses to both alcohol and thiamine deficiency that renders the alcoholic so very sensitive to damage in these important brain regions. To blame thiamine alone could be but a partial view of the problem.

Heterogeneity

The next principle to be considered is perhaps the most platitudinous of all, but it certainly deserves close attention. With regard to vulnerability to brain damage alcoholics are remarkably heterogeneous. The soil as well as the seed is clearly relevant.

Thus it was always apparent from the CT studies that there was great variability, some patients showing brain shrinkage while others, remarkably, were unaffected. This has been noted in virtually all reported studies – about a third of subjects seem resistant to this development despite drinking histories of equivalent severity to the remainder. Psychometric surveys have arrived at the same general conclusion: we must not be misled by group mean values alone since performance on tests varies widely within any alcoholic group. Parsons (1987), in a valuable review, concludes that a substantial number of alcoholic subjects in most surveys have remained intellectually intact.

Vulnerability to the Wernicke-Korsakoff syndrome also appears to be highly variable, for reasons that are largely unknown. A young woman was reported recently who developed Wernicke's syndrome due to alcoholism at the age of 18 (Turner *et al*, 1989). Even from place to place its incidence appears to be remarkably uneven (Price & Kerr, 1985). In everyday clinical practice we encounter patients who amazingly escape this complication despite neglecting their diets and drinking enormous quantities. Numerous factors are likely to be involved – the beverage chosen, its thiamine content, patterns of drinking, and the patterns of dietary neglect. The context of alcohol abuse is itself very heterogeneous.

However, there may be an important additional factor, by way of personal susceptibility. This has long been suspected and there have been some inquiries into its possible biochemical basis. Thiamine is important in relation to several key enzyme systems of the body and brain. It is first phosphorylated to thiamine pyrophosphate (TPP) which acts as a coenzyme, that is, it combines with protein molecules to form the effective enzyme system. This applies, for example, to enzymes such as transketolase, the pyruvate dehydrogenase complex, and the α-ketoglutarate dehydrogenase complex, all intimately concerned with brain glucose metabolism and with the synthesis of neurotransmitters. The question arises whether the person vulnerable to thiamine deficiency could have an *inborn abnormality* by way of reduced affinity between TPP and the enzymes with which it must combine.

Kaczmarek & Nixon (1983) and Pratt *et al* (1985) have shown that transketolase is heterogeneous, and that some variants differ in their affinity for TPP (Greenwood *et al*, 1984). Blass & Gibson (1975) and Nixon (1984) have identified variants seemingly specific to Korsakoff patients. It has been hard, however, to exclude the possibility that such variants are the *product* of damage to the system rather than genetically transmitted defects.

The new molecular genetics may ultimately be used to clarify the position. Armed with genetic probes, constructed by sequencing the amino acids of the isoenzyme variants, we may one day be able to study the inheritance of vulnerability to thiamine depletion. Other enzymes additional to transketolase could warrant such attention in relation to thiamine deficiency. And the enzymes involved in the detoxification and metabolism of alcohol itself could repay equivalent study. Looking to the future, perhaps not so very far distant, we may be able to identify those individuals who warrant particularly close surveillance because of their inherited susceptibilities.

A hypothesis

It is traditional, in seeking scientific progress, to attempt to construct a hypothesis. As an important step at this point it may therefore be valuable to draw various strands of evidence together and see where they may lead. In parts the hypothesis will admittedly be built on rather shaky ground but may yet serve a useful purpose.

We have noted that the alcoholic lives at risk of two major forms of cerebral insult, the first due to alcohol neurotoxicity and the second due to thiamine deficiency. This much can scarcely be doubted. We have seen that each may often leave its mark well before we suspect pathological developments, yet contributing to the alcoholic's deficits. Both are potentially remediable, in the early stages at least, the first by prolonged abstinence and the second by thiamine replacement.

Alcohol neurotoxicity, the hypothesis proposes, affects both the cortex and the subcortical structures. The cortical effects are detectable during life by shrinkage on CT scan; the subcortical effects on the basal brain regions largely escape such detection. Thiamine depletion has a marked and well attested effect on these same basal regions. They are therefore at special risk since they are vulnerable to both types of damage:

$$\begin{tikzcd}
\text{Alcohol neurotoxicity} \arrow[r] \arrow[dr] & \text{Cerebral cortex} \\
\text{Thiamine depletion} \arrow[r] & \text{Basal brain regions}
\end{tikzcd}$$

Individual alcoholic patients, the hypothesis continues will differ greatly in their susceptibility to each type of cerebral insult, perhaps largely by virtue of genetically controlled biochemical factors. Such factors will couple in an important and sometimes decisive fashion with patterns of alcohol abuse and dietary neglect in leading to the damage sustained.

Thus the individual who has heightened susceptibility to alcohol toxicity alone will live at hazard of varying degrees of cerebral shrinkage and cognitive impairment, which will tend to recede with abstinence:

$$\begin{tikzcd}
\text{Increased susceptibility to alcohol neurotoxicity alone} \arrow[r] & \text{Cerebral shrinkage and cognitive impairment, tending to recede with abstinence}
\end{tikzcd}$$

The individual who is vulnerable to thiamine deficiency alone will tend to develop a mild and transient Wernicke–Korsakoff state; he may also sometimes feature among that group of patients who show thiamine-dependent pathology in the brain at autopsy when there had been little to point towards it during life:

$$\begin{tikzcd}
\text{Increased susceptibility to thiamine deficiency alone} \arrow[r] & \text{Mild and transient Wernicke–Korsakoff state}
\end{tikzcd}$$

But some individuals relatively rare, will have the dual vulnerability:

$$\begin{tikzcd}
\text{Increased susceptibility to alcohol neurotoxicity } \textit{and} \text{ thiamine deficiency} \arrow[r] & \text{Chronic resistant Korsakoff's syndrome}
\end{tikzcd}$$

These people will be at very special risk. The combined effects of heightened susceptibility both to alcohol and to thiamine deficiency will stand to damage the basal brain regions severely. Thiamine deficiency will in effect supervene on brain regions which have already been compromised in some degree. Structural brain changes will more readily ensue, and the Korsakoff state will prove resistant to thiamine replacement.

This may be why chronic Korsakoff's syndrome is relatively rare, and why not every alcoholic is at equivalent risk. This may be why our own Korsakoff sample showed such prominent enlargement of the lateral ventricular system and the interhemispheric fissures. These were the markers, on neuroimaging, of their increased vulnerability to alcohol neurotoxicity, a vulnerability which had also primed the subcortical structures to react severely to thiamine deficiency.

One must add, for the sake of completeness, the important qualifying conception that brain biochemical changes will follow from the damage sustained by the nuclei at the base of the brain. How far, and by what routes, this may sometimes lead to a circumscribed Korsakoff state, and sometimes to more global dementia, must remain conjectural. Age factors, at least, are likely to play a role, since with ageing the cholinergic system becomes more vulnerable. Further knowledge of the organisation of brain biochemical mechanisms clearly stands to be relevant to understanding of the alcoholic brain.

Therapeutic implications

When treating an alcoholic patient we must recognise that even when not intoxicated he is often struggling to combat his dependence with a brain that is malfunctioning. In addition to cognitive deficits there are disorders of affect, motivation and compliance which will derive just as surely from the damage he has sustained. To the extent that we can remedy such cerebral abnormalities, or delay their development, we may be intervening in a beneficial manner.

Firstly, we must be aware of the need to aim at prolonged abstinence in certain patients before attempting the full processes of rehabilitation. Some patients are too compromised to benefit from group or individual psychotherapy until a considerable time has gone by. Psychometric screening would appear to warrant wider employment, even in busy treatment units.

Secondly, there may be more leeway than we think, and in far greater numbers of patients, to prevent the build-up of covert thiamine deficiency. Vitamin supplementation should perhaps be more widely practised; the thorny question of thiamine supplementation of beverages (Finlay-Jones, 1986) deserves to remain on the agenda.

Thirdly, we may have more room than we realise for the manipulation of neurotransmitter systems, particularly in the overtly damaged alcoholic. Pharmacological remediation, or amelioration, of the deficits of Korsakoff's syndrome should remain a valid goal. It is perhaps no more than the current tide of research fashion which has led to such close (and valuable) attention to Alzheimer's dementia, while biochemical and pharmacological research into alcoholic brain damage remains so little studied.

Lastly, the detection of genetic vulnerability to brain damage in the alcoholic subject may ultimately be a realistic possibility. Disorders of enzyme structure and function are ideal candidates for the molecular genetic approach. We must perhaps wait patiently for some serendipitous discovery to catalyse the turning of attention by this latest of our laboratory disciplines to the alcoholic brain.

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Lecture

The Social Psychiatry of Later Life

A. S. HENDERSON

Throughout the two million years of human existence, it is without precedent that the elderly should now constitute such a large proportion of the world's population. Although it is already trite to emphasise this remarkable change in the global population structure, the increase is nothing less than an awesome imperative for public health as a socially responsible science. By the year 2025 AD, the United Kingdom will have 14 million persons aged over 60, which would be 26% of the population. Just under two million of them will be over 80 years old (United Nations, 1985). In Australia and New Zealand, 24.8% of the population will be aged 60 years or more. About one million Australians will be 80 years or over. West Germany will have 2.4 million aged over 80, the USA 7.7 million. The People's Republic of China will have 19.3% of its 1.5 billion people who will be aged over 60, and over 25 million will be over 80 years.

These simple figures carry profound implications for people's personal lives, their work pattern, leisure and housing, and for national economies, and their health and social services. The demographic change is itself a sufficient reason for psychiatry to prepare itself. This preparation will have to be in knowledge, education, manpower, and policy. Social psychiatry has already yielded knowledge complementary to some of the laboratory advances in the field of ageing, such as the neurochemistry and molecular biology of dementia. A reasonable body of knowledge has accumulated about the distribution of mental disorders in the elderly, and about social factors influencing that distribution. But in the face of both demographic changes and rapid progress in the neurosciences, social psychiatry has now come to have an enhanced responsibility. This is largely because of what it deals with. A definition of the subject can be offered as follows:

> "Social psychiatry is concerned with the relationship between disorders of the mind and the human environment. It studies the forces which act at the interface between individuals and those around them; and which may contribute to the onset, or influence the course of mental disorders." (Henderson, 1988*a*, p. 1)

In an important essay in the *Handbook of Social Psychiatry*, Sartorius (1988) has told us that "social psychiatry will disappear and it is likely that the world will be a slightly better place without it". His argument is that social psychiatry has done much for the mentally ill, and its principles should now be integrated within health care as a whole. While this would be a major achievement, there would also be the danger that, through its dispersion, no one would specialise in social psychiatry, and think about its issues in depth.

To examine what knowledge social psychiatry currently has available, and to identify the areas of ignorance, it is useful to have a system to organise one's thinking. For this, I have found it helpful to use a matrix (Henderson, 1988*a*, p. 31). The columns are defined by the main diagnostic groups, and the rows by variables relevant to social psychiatry and epidemiology. In this lecture, I deal mainly with dementia and depression. But against what social variables are these to be considered? The rows start with sociodemographic factors. Next are experiential variables such as life events, social support, social stimulation, and being physically ill or disabled. There are physical exposures to consider: diet, medication, or toxins in the environment. Macrosocial forces such as economic changes or shifts in values may have an impact on older persons. Next there are the social consequences of mental disorders in the elderly — on the person, on the family, and on the community. This takes us to consider services, asking what proportion of the elderly with mental disorders reach general practice, and how appropriate are the services obtained there or in hospitals and institutions. In social psychiatry certain social treatments have evolved which seem to be highly relevant in the elderly. Lastly, there is the question of prevention, which Gruenberg (1990) reminds us is the ultimate service of epidemiology. In this way, cell by cell, the matrix provides a framework for the social psychiatry of later life.

Eli Lilly Lecture, Royal College of Psychiatrists, London, 24 January 1989

Dementia

Knowledge about dementia as it occurs in the general population comes principally from epidemiological studies. These surveys have been conducted with the assumption that individuals in the populations examined can easily be placed into two categories: cases of dementia and normals. But in his excellent review of epidemiological research on dementia, Cooper (1988) rightly points out that "it seems quite plausible that clinical dementia in the elderly represents one end of a spectrum of cognitive decline". Epidemiological studies should examine the distribution of this continuum, rather than focus only on persons at the extreme end. Furthermore, the cut-off point selected on that continuum has varied considerably between investigators.

There have now been over 50 surveys of dementia in general populations. Jorm *et al* (1987) carried out an integrative review of 47 prevalence studies conducted between 1945 and 1985. Four main findings emerged. First, the prevalence of dementia increased exponentially with age, with a doubling of rates every 5.1 years, at least up to the age of 95 years. Second, the prevalence rates varied greatly between surveys, leading the authors to declare that, "It may be that true regional differences do exist, but their magnitude is impossible to assess with existing data, because of the many methodological differences between studies." A third finding was that dementia occurred at an equal rate in men and in women, but when senile dementia of Alzheimer type (SDAT) was considered as a specific type, women had higher rates. A subsequent analysis of incidence data showed no sex difference, leading Jorm (1990) to conclude that there is differential survival. Fourth, the prevalence rates of SDAT and of multi-infarct dementia (MID) were significantly different between regions: the Japanese and Soviet surveys (e.g. Gavrilova, 1977; Karasawa *et al*, 1982; Hasegawa *et al*, 1984) reported higher rates for MID than for SDAT, while nearly all the other studies reported either no difference or higher rates for SDAT. This is not an observation to pass over lightly. If it were demonstrated that there are true differences in the prevalence of Alzheimer's disease between world regions, this would be a clue to aetiology of some significance. Among a number of competing interpretations would be that there is differential exposure to some causal agent.

Regarding the Russian situation, Shefer (1987) has recently argued in the Soviet *Journal of Neuropathology and Psychiatry* that the general assumption among Soviet clinicians that vascular dementia is more common is false. On the basis of post-mortem examinations of the brains of 714 elderly persons, he concluded that there is "a marked hyperdiagnosis of hypophrenias of vascular genesis at the expense of AD and senile dementia whose rates are obviously underreported . . . ". We can conclude that the Russian rates are probably similar to the European.

Recently, more non-Western studies of prevalence have come to our notice. Working with a sample of 12 000 households spread over 12 regions of China, Zhao (1986) studied 4619 persons aged over 60 years. The prevalence of MID and SDAT were 3.3% and 2.4% respectively. A particularly notable study has recently been completed by Li *et al* (1989) in the elderly of Beijing. The Mini-Mental State Examination (MMSE; Folstein *et al*, 1975) was used in phase 1, with a cut-off score of 17. In phase 2, involving clinical assessment, rates for MID were higher than for primary progressive dementia. All those suffering from dementia were cared for by relatives in their own homes.

For Japan, a number of recently published studies have been brought to our attention by Professor K. Hasegawa. Two examples are the surveys by Motohiro (1985) in Fukuoka City and Shibayama *et al* (1986) in Nagoya. In both of these, the notable features were the large samples, the high contact rates, the use of highly trained personnel, usually in a two-phase design, the very reasonable values of 4–6% found for overall prevalence, and the consistent excess of MID over SDAT. Having examined the diagnostic criteria used by Hasegawa *et al*, I am persuaded that the rates for AD and MID used in their studies are likely to be largely free of diagnostic artifact, and are almost certainly valid estimates.

Looking at this wealth of epidemiological data from many parts of the world, one is tempted to conclude that a definitive cross-national field survey would be well worth conducting, to settle the question of variable prevalence. This is now recurrently advocated in the course of international meetings on dementia. The problems are formidable, although not necessarily insuperable. Here are just some of the requirements: the centres should have at least some common aims; the samples should be comparable in age structure and in the inclusion of persons living in institutions; case finding and case definition must be by the same standards and method; if prevalence is being estimated, the disorder should be known to last the same length of time, on average, in each population; and throughout the planning, field work and analysis, the investigators have to maintain a working relationship. These requirements often prove difficult to meet.

Twenty-five years ago, in the Edinburgh MRC Unit, Kessel (1965) wrote a spirited paper with the title "Are international comparisons timely?" He persuaded the reader they were not, concluding that "it is the field survey carried out internationally that provides the least return for most effort". This was written before the successful US–UK Cross-National Diagnostic Project (Copeland *et al*, 1975, 1987; Gurland *et al*, 1983), which partly contradicts Kessel's view. But I believe this is because that study involved only two cities, both of which have a common language and basically the same Western culture. International surveys of dementia in the future, if they are to aim at aetiological factors, should likewise be carried out between relatively homogeneous settings, at least until biological screening tests for AD and MID are available for epidemiological use.

Is dementia becoming more or less common? As we all recognise with admiration, a study of the incidence of mental disorders has been conducted in Sweden by Hagnell and his colleagues for many years. This has included rates for both senile and multi-infarct dementia. A preliminary report (Hagnell *et al*, 1983) suggested that the rates for both dementias had fallen over the 25 years of observation, but a further analysis by Rorsman *et al* (1986), which included newcomers to the town, found no change. This is not to say that secular change in the incidence of dementias does not exist, but it is very laborious and expensive to investigate it.

For Alzheimer's disease, a critical review can now be made of all proposed as risk factors (Henderson, 1988*b*). Very few of the proposed factors are securely established, such as age itself, gender, and, for the familial form, a history of dementia in a first-degree relative. Others are far from certain and some, perhaps the most interesting, are only hypotheses. This second group includes education, ethnicity, Down's syndrome in relatives, vascular dementia, thyroid disease, head injury, previous depressive illness, urban versus rural living, geographic variation, smoking, analgesics, aluminium, organic solvents, and vibrating tools. McWhorter & White (1987) studied the Framingham cohort ($n=4236$) to examine factors associated with the later development of primary progressive dementia. They found an increased risk for persons with a previous history of diabetes, physical inactivity, heart failure, or having less than one bowel movement a day.

One hypothesis has been put forward which would go some way to unifying a number of the proposed risk factors (Henderson, 1988*b*): what features, if any, were shared by some of the proposed risk factors? Abnormal peroxidation with the formation of free oxygen radicals is known to increase with age, with ischaemia, trauma, phenacetin, and organic solvents. Pursuit of the free radical and abnormal peroxidation hypothesis has been supported by Beyreuther *et al* (1988) and by Deary & Whalley (1988), although the reservations of Dormandy (1988) have also to be considered. Epidemiological studies now need to seek opportunities to test the hypothesis. This resolves itself into asking the right questions about persons who may have been exposed to free radical attack.

In studies currently under way in several centres, there will be an opportunity to look carefully at two attractive sociodemographic hypotheses: that there is a gradient in prevalence by social class or education for AD, net of artifact in case finding; and that there are true regional differences in prevalence within a country. While the educational gradient may correctly be attributed to artifact induced by the tests themselves or the persons using them (Gurland, 1981), an alternative hypothesis is that part of the difference is real, and that poor education or low social class is associated with something which contributes to the onset of AD. Such an association would be of considerable epidemiological significance. For differences within a geographic region, there are a number of positive reports (e.g. Sulkava *et al*, 1985), but none is in any way conclusive. Martyn & Pippard (1988) showed in an elegant analysis that death certificates for dementia were not a satisfactory method for investigating possible regional differences in prevalence. Using mortality rates for each of the seven Australian states, we found that the apparently high prevalence in one of them was attributable to the preferences of one practitioner in writing death certificates (Jorm & Henderson, 1990).

To make further headway in identifying risk factors, some investigators are now working with special populations such as twins, population isolates, or élites. In collaboration with neurological colleagues in Sydney (Professor G. A. Broe and Dr H. Creasey), we have used the Australian NH & MRC Twin Registry to identify three pairs of elderly monozygotic twins discordant for AD (Creasey *et al*, 1989). We made an exhaustive search of their earlier lives in respect of health, life-style, and possible exposures. No consistent differences were found between pairs. Yet the fact that monozygotic twins discordant for AD do exist strongly suggests that environmental or experiential factors play a major role in the onset, if not in the aetiology of sporadic AD.

Carers of persons with dementia

An issue of great social relevance in the management of persons with dementia is what can be done for the carers. As we know, the overwhelming majority are women, receiving little assistance from others; and this is called community care. In a classic paper in the *Lancet* 30 years ago, Macmillan (1960) proposed that increasing strain on a family reaches a critical point at which they may reject the care of the patient, and do so irreversibly. This gave emphasis to the desirability of early intervention. Grad & Sainsbury (1968) and Bergmann *et al* (1978) both found that those families caring for an elderly relative were not receiving the advice and relief from stress which they needed. Indisputable evidence has now accrued that those who care for relatives with dementia endure considerable stress (e.g. Gilleard *et al*, 1984; Wells & Jorm, 1987; Gilleard, 1987). Help lies in the admission of the old person to care, day care, respite care, or additional domiciliary services. There is now a growing body of information on how well each of these works. In Melbourne, Wells & Jorm (1987) have reported a small randomised controlled trial of admission to a special nursing home for dementia sufferers. The high levels of psychological symptoms in care-givers fell dramatically in those whose relative was admitted, and the level stayed low. Cognitive function in both the nursing home and control cases of dementia continued to decline, although with little difference between the two groups. Full admission, it seems, brings relief to the carer, but this can be given only to a minority.

The impressive review by Morris *et al* (1988) helps us to look more closely at what being a carer means, and what goes on in their minds. These authors place emphasis on the importance of cognitions and attributional style in the carer, who may often experience a loss of control over several aspects of her daily life. It is information of this nature that puts us in a stronger position to supply those elusive commodities which we call support, while not really understanding what this term subsumes.

Much more needs to be learned about what actually helps carers. There is often a compulsive insistence by policy makers on community retention. Day care is a much-used compromise; and it is supposed to help both the sufferer and the family. That is a remarkable assumption, which needs to be looked at more closely. In a collaborative study in four areas of Australia, Wells *et al* (1990) found that the high level of psychological symptoms in care-givers was *not* significantly reduced by day care for their elderly relatives. Full-time admission of their relative to a nursing home did reduce distress. The investigators concluded that day centres may provide too few hours of relief in the 168-hour week to benefit the mental health of the care-givers. If such findings are confirmed, the implications for policy and for research needs are not trivial.

Depression

Affective disorder in the elderly tends at present to be eclipsed by concern with dementia. Information about the occurrence of depressive disorders in the elderly can be assembled from different epidemiological levels. It is field surveys which provide the only possible opportunity to examine the social and other correlates of depressive disorder in the aged, free of those factors which determine who reaches medical or institutional care. There have been at least 25 such surveys conducted in the last 40 years. As with dementia, the estimates reported are highly variable, attributable to differences in methods and in criteria. Under such conditions, it is not usually possible to look for regional differences in rates as a clue to aetiology. An exception has occurred where the same instrument and the same diagnostic algorithms have been used by field workers with similar training. Copeland *et al* (1987) examined the data on the elderly from the US–UK Cross-National Diagnostic Project (Gurland *et al*, 1983). They found that, unlike dementia, depression was not significantly different in its prevalence between London and New York. This is an engaging observation if we bear in mind the different social environments for the elderly in the two cities. Geographic differences in the prevalence of depression in the elderly may exist, but they have not yet been demonstrated.

Epidemiological data can be used to answer the next question: aside from selective access to services, is there more or less depression in the elderly than in younger adults? For various reasons, it is intuitively reasonable to expect higher rates in the elderly. The theoretical predictions of Erikson (1961), which presumably are universally applicable, are that the elderly either achieve ego integrity or despair. There are also losses in health, in personal networks, and in social status, which is jargon meaning that one is no longer valued so highly as a person, but instead becomes an encumbrance. If social circumstances influence the onset of depression or its duration, the elderly might be expected to carry higher prevalence rates.

Gurland (1976) held that the majority of definite depressive disorders appear before the age of 65 years. He quoted Silverman (1968), who said that depression "reaches its greatest frequency in the middle years, and may decline somewhat in the later years of life". Eaton & Kessler (1981) found a strong linear tendency for the young to be more depressed than the elderly, after controlling for variables such as marital status and education. More recently, interesting data on depression and age have come from the five sites of the Epidemiologic Catchment Area (ECA) studies. Robins *et al* (1984) reported strikingly lower rates for *lifetime* major depressive episode in the elderly than in other ages, in New Haven, Baltimore, and St Louis, although the validity of such self-reported lifetime morbidity has been questioned by Parker (1987). Myers *et al* (1984) found that the *six-month* prevalence of major depression and dysthymia was also lower in the elderly in all three sites. At the fourth site, the Piedmont of North Carolina, Blazer *et al* (1987) found the rates in close agreement with the values obtained for the elderly in the other three sites. The age-specific one-month prevalence of affective disorder has now been reported by Regier *et al* (1988) for all five sites of the ECA study. Persons aged 65 years and over had a rate of 2.5%, the lowest across all age groups, and in marked contrast to the rate of 6.4% in persons aged 24–44 years. Women in this latter age group had a one-month prevalence of 8.2%. The conclusion is that on the Diagnostic Interview Schedule (Robins *et al*, 1981) and using DSM-III criteria for depressive disorders (American Psychiatric Association, 1980), the elderly in the community have lower six-month prevalence rates than younger adults, and that this is probably not an artifact of reporting. In this same lecture series Klerman (1988) proposed from several sources of evidence that there has been a cohort-bound effect causing an increase in depression in young adults. The elderly may have been spared this increase.

The contribution of social factors to aetiology

Clinicians know better than to attend exclusively to social factors when trying to understand the genesis of a patient's illness. But social psychiatry seems repeatedly to have assessed only environmental factors in individuals' current or earlier lives, with the expectation of finding substantial causal effects (Henderson, 1988*a*, p. 174). When Post (1972) described his clinical series of 92 elderly persons with depression, he concluded that "the phenomena observed in every depressive attack are uniquely shaped by the constitutional status of the patient at the time of the attack, by the emotional significance of preceding events, and by existing strengths and weaknesses of his personality structure". Is it likely to be different in other clinical series? Is it likely to be different in general population surveys of the elderly? In her San Francisco studies, Lowenthal (1965) found that those in psychiatric care in hospital, although not all suffering from depression, showed evidence of greater social withdrawal since about the age of 50 years; by contrast, persons who lived alone all their lives were evidently no more prone to mental illness in old age than anyone else. A close relationship was associated with good mental health; and late-developing isolation, due to loss, was associated with the onset of mental disorder.

A study directed expressly at the social origins of depression in old age was carried out in London by Murphy (1982). One hundred referred patients and 19 depressed persons from a community sample were compared with 200 mentally healthy community residents, matched for age and sex. She found that depression was closely associated with adverse events involving threat or loss, just as Brown and his colleagues have consistently found in younger adult women (Brown & Harris, 1978). Murphy noted that for the elderly, many losses are irretrievably hopeless: that is, they have a quality of finality about them. On the other hand, it is likely that the elderly actually have fewer events, on average, and fewer severe events, than younger adults. Furthermore, they report less subjective distress. In the Canberra survey, we found a correlation of −0.46 for the frequency of events and age, and −0.33 for distress scores (Henderson *et al*, 1981). Nevertheless, the onset of depression does seem to be associated with adverse events. Murphy found that the risk of developing a depression in the face of a severe adverse event was significantly higher in the elderly who had no confidant. The study confirmed the now well established association between physical illness and the onset of depression (Roth & Kay, 1956; Kay & Bergmann, 1966). Murphy (1982) remarked how "the majority of older persons who are suffering from social problems, adverse life events, and poor health remain remarkably cheerful; and it is only those who lack a confiding relationship who appear to be vulnerable to depression in the face of these problems". She proposed that a confidant can act as a buffer, and suggested that the essential protective quality is the capacity for intimacy, which is one aspect of pre-morbid personality. This, she proposed, is a "*major vulnerability factor*" (her italics). Murphy also reported that 37% had had a previous episode of depression in younger life, and that in 15% it genuinely had no precipitating event, and no evidence of adversity or social stress. But she concluded that across her whole sample of cases, social factors appeared to be as important in depression in the elderly as in younger persons. The merit of this work is that it set out to find social factors, but attended also to psychological and biological variables, not as separate domains, but in interaction, just as Post would have advocated.

In Hobart, Australia, D. W. K. Kay and our unit conducted a cross-sectional survey of dementia, depression, and social support in the elderly (Kay *et al*, 1985; Henderson *et al*, 1986*a*,*b*). The elderly as a group had fewer social relationships than younger adults, but were more content with what they did have. The Hobart elderly with depression reported having markedly less social interaction than the mentally healthy, but did not complain about this. They did not lack intimate ties. We then went on to examine the mental health and social environment of elderly persons who live alone (Henderson *et al*, 1986*b*). They had markedly fewer close relationships, as expected, but as much general social interaction as other elderly persons. They experienced much more loneliness. Although they had no greater a prevalence of depression or dementia by DSM-III criteria, they did have more neurotic depressive symptoms, including loneliness. We concluded that, in that community, the elderly who live alone could not be said to be at increased risk for mental disorders. They were certainly not socially isolated. Cooper *et al* (1989) have recently investigated a sample of the elderly in Mannheim, West Germany, also finding no excess of morbidity in those living alone, or in those with few social contacts. Nevertheless, as many as 27% of their sample had multiple indicators of social isolation, and were to be regarded as prospectively at high risk for admission to long-term care.

In epidemiological work on social support in later life, there have been some recent developments sufficient to evoke a review paper on social relationships and health by House *et al* (1988), in *Science*. The interest goes beyond psychiatry, because it now seems well established that persons who have plentiful social relationships, not necessarily affectionately intimate ones, have better physical and mental health, and a lower mortality rate. This holds after allowing for confounding by health-related behaviour and access to services. A recent illustration is the finding by Cooper *et al* (1989) the mortality in institutional residents in Mannheim can be predicted in part by the frequency with which they have been visited. The term 'social support' is a weak one, in that the metaphor of support does not do justice to what Weiss (1974) so usefully called "the provisions of social relationships". What is needed now is to determine which of these provisions have an effect on the brain, and how that effect is mediated.

Care-eliciting behaviour in later life

The concept of 'care-eliciting behaviour' (Henderson, 1974, 1977) is highly relevant to the care of the elderly. The term is derived from animal ethology (Scott, 1969) to describe a spectrum of behaviour which elicits caring responses by others of the same species. Many care-eliciting types of behaviour are both universal and highly adaptive, such as the crying of an infant. Konner (1972), referring to this sound in the Zhun-twasi hunter-gatherers, said, "It is the same intensely unpleasant sound, the one one wants to hear stopped immediately." But there is also adult behaviour which elicits care from the person's immediate social environment with an urgency similar to the infant's cry. I suggest that these are distributed along a dimension of how disruptive they are to the person or to others.

An individual of any age who perceives a lack of care and concern from others may engage in care-eliciting behaviour. The comfortably-off middle-class wife who lacks affection in her marriage may work her way up the care-eliciting dimension of behaviour until she engages in shop-lifting. This is carried out in a state which can conveniently be called 'anophelia' (from ωφελεια, 'ophelia', as in the woman's name, meaning care or help, that which is accorded to a person in distress) (Henderson *et al*, 1981, p. 15). Now the elderly must certainly experience anophelia, and may cause themselves and their families distress by their eliciting care. It is also a state which occurs in institutional settings. Abnormal care eliciting can take the form of unreasonable requests for care and attention, but it can also be of one particular type relevant to general or geriatric practice. This is where individuals either have or believe they have a medical disorder, and act inappropriately. Here we have the phenomenon collectively called "abnormal illness behaviour" by Pilowsky (1969, 1988). It is defined as the persistence of an inappropriate mode of perceiving, evaluating, and acting in relation to one's health, despite having had an accurate explanation from a doctor (see Pilowsky, 1988, p. 306). Physical symptoms can be used as instruments to elicit care and attention from others. Since the elderly have an increased level of physical morbidity, they have ready access to what Pilowsky calls the "currency" to be used and negotiated in the course of personal relationships.

General practice

If general practice is the linchpin of effective services for the elderly, it is a matter of central importance to have information on three issues: (a) the proportion of all elderly persons with psychiatric disorders who go forward to seek help from general practitioners (GPs) — the so-called first filter of Goldberg & Huxley (1980); (b) the proportion of these who are correctly recognised by their GP (the second filter); and (c) the adequacy of the treatment or management given. Such information could be put to good use in helping GPs achieve the highly effective service for their elderly patients for which they are so well placed.

It is likely that the elderly under-consult for psychiatric problems, just as they do for physical complaints. They may attribute their depressive symptoms to their years and circumstances, not expecting that a doctor would be able to help. Likewise, a decline in memory and thinking in an elderly person may not always prompt a relative to seek medical advice. The next issue is the performance of GPs in recognising morbidity, particularly depressive disorders and early cognitive failure or mild dementia. The much-cited study by Williamson *et al* (1964) in the Edinburgh area showed that only about one-fifth of cases of depression or dementia were known about by the doctor. Parsons (1965) found that Swansea GPs were aware of about half of those elderly persons who were markedly handicapped by a psychiatric condition at the time of the visit. In Upper Bavaria, Weyerer (1983) found that the elderly had a much higher use of general practice services than younger adults, and that the GPs performed very satisfactorily in recognising psychiatric disorders. Referral to psychiatrists, though, was at a much lower rate than for younger adults. Philp & Young (1988) have reported most encouraging findings in their audit of a primary care team's knowledge of the existence of dementia in their elderly clientele. In a six-partner training practice in Clackmannanshire, they found that no person with symptomatic dementia was not known to the practice to be so impaired. But the GPs and practice nurse did not know about many of those with mild cognitive impairment. Brayne & Calloway (1990), in their survey of rural women, found high rates for GP recognition of dementia, while in the Hughes Hall Project, O'Connor *et al* (1988) found that the GPs correctly identified dementia as at least a possibility in 121 of the 208 cases of dementia in the study. Both they and community nurses mistakenly identified as demented several persons with functional disorders, particularly depression.

We have recently analysed the use of private psychiatric services by the whole of the population in Australia (Jorm & Henderson, 1990), where about 77% of psychiatric services are provided by private practice (Andrews & Hadzi-Pavlovic, 1988). Again, the elderly use such services much less than other adults. The length of each session is also much briefer than for younger adults. It does seem that when GPs recognise a psychiatric disorder in elderly patients, they are much less likely to refer them to a psychiatrist. A relatively encouraging finding by Macdonald (1986) was that GPs missed few cases of depression, although they used antidepressants seldomly and had low rates of psychiatric referral.

Because of the central role of GPs and their teams in the management of mental disorders in the elderly, I would strongly advocate that special initiatives now be introduced for improving diagnosis, and for improving treatment standards. Without such action, better clinical management together with longer retention within the community will be hard to achieve.

Preparing for the future

In only 36 years' time, a quarter of the population of Europe will be 60 years or over. Demographers do not give us information on when this remarkable expansion of the elderly will stop. They describe what they call the humanitarian consequences of the predicted increase (United Nations, 1985). But this increase will be accompanied by awesome changes within psychiatry itself. The recent meeting in New York to consider these noted the striking advances now being achieved in the understanding of brain function (see *Science*, 1988, p. 1013). This places an added responsibility on psychiatry to integrate such advances with social and cultural factors. While this requirement will hold across all age groups, it does so nowhere more strongly than with the elderly. The 'neurotechnologies' now being developed are likely to assist in early diagnosis of dementia, in its treatment (including brain transplants) and, through molecular biology, in its prevention. But epidemiology tells us how formidable the number of candidates will be, in relation to resources. In developing countries, where the greatest increase in the elderly is occurring, it is hard to accept that the large number of demented people will personally benefit from these emerging neurotechnologies. For the whole of the human population, dementia is unlikely to go away during the next few decades, or even to diminish in prevalence. Meanwhile, by far the most effective resource in its management is the family. Both clinicians and governments know this.

For the present, the immediate agenda for the social psychiatry of the elderly is clear. There is the platform of knowledge currently available across the matrix of issues to which I referred earlier; there are those problems which have scientific appeal; and there are the imperatives of indisputable public health need. In the light of this, I suggest that the social psychiatry of later life has now to harness its resources to tackle the following as a basic agenda: (a) a continued search by epidemiological methods for clues to the aetiology of Alzheimer's disease; (b) the development of bold and innovative means for assisting families to care for a relative with dementia; (c) studies of the contribution of general practice to the care of mental disorders in the elderly, and how to improve that contribution; and (d) an intensive, theory-driven investigation of the social environment in mental disorders of the elderly, and in the promotion of their well-being. In this, the elucidation of the social support hypothesis is particularly enticing, including its link with mortality. These are matters which we do have the intellectual resources and instruments to tackle. They carry great social relevance for our communities, as these are and as these will shortly become. They are also scientifically attractive questions. In these tasks, social psychiatry will continue its contribution to public health.

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Relatives and Patients as Partners in the Management of Schizophrenia

The Development of a Service Model

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Considerable advances have been made in the family management of schizophrenia but there remains a major challenge for the psychiatric services to integrate these innovations into clinical practice. A number of important issues need to be considered in developing routine clinical services: the problem of engaging families in a therapeutic programme; the utility of the concept of 'expressed emotion'; and procedures for clinical practice. The latter include the needs of low-EE families; maintaining quality of intervention in a clinical context; responding to the multiplicity of needs of the patient and family; and integrating family interventions with ongoing rehabilitation practice. A model of service provision is described.

The last decade has witnessed a renewed interest in the family of the individual with schizophrenia, in particular with regard to the impact which the family may have on the course of the disorder itself. This revival stems directly from research into 'expressed emotion' (EE), which has generated a burgeoning literature centring on issues of prediction, validation, and treatment (Leff & Vaughn, 1985). Six family intervention studies have been published (Goldstein & Kopeiken, 1981; Leff *et al*, 1982, 1989; Kottgen *et al*, 1984; Falloon *et al*, 1985; Hogarty *et al*, 1986; Tarrier *et al*, 1988) (Table I), and although much remains to be done to explore the limits of their internal and external validity (Hogarty *et al*, 1986) the time is now overdue when the fruits of this research should influence clinical practice (Kuipers & Bebbington, 1985). This is particularly apposite in view of the increasing trend towards community-based mental health services.

New drug treatments are relatively easily incorporated into clinical practice; however, psychosocial interventions represent complex and time-consuming procedures which require careful planning of mental health resources when attempting to integrate them into existing psychiatric management. Many questions are therefore raised when considering the possibility of routine service to families. Can they be provided efficiently yet effectively? Who should receive the intervention and what form should it take? Should the intervention be provided through referral to 'specialists' or more fully incorporated into the existing treatment system? Who has the requisite expertise to deliver the interventions? Can they be afforded?

TABLE I
Family intervention studies[1]*: relapse rates*

Study	n	*Follow-up* *9 or 12 months*	*24 months*
Goldstein & Kopeikin (1981)[2]			
family intervention	25	0	—
routine treatment	28	16	—
Leff *et al* (1982, 1989)			
family intervention	12	8	20
routine treatment	12	50	78
family therapy	12	8	—
relatives group	6	17	—
Kottgen *et al* (1984)			
family intervention	15	33	—
control (high-EE)	14	43	—
control (low-EE)	20	20	—
Falloon *et al* (1985)			
family intervention	18	6	17
individual intervention	18	44	83
Hogarty *et al* (1986)			
family intervention	21	19	—
social skill training	20	20	—
combined intervention	20	0	—
control	17	41	—
Tarrier *et al* (1988)			
family intervention	25	12	33
education only	14	43	57
routine treatment	15	53	60

1. All subjects in these studies were in receipt of neuroleptic medication.
2. The follow-up period for this study was six months.

Based on a paper presented to the World Congress in Behaviour Therapy, Edinburgh, 1988.

With these issues in mind, the West Birmingham Health Authority in conjunction with the University of Birmingham mounted a research and development programme in order to develop, initiate, and evaluate services to individuals with schizophrenia and their families. This represents one of the first attempts to integrate these advances in treatment into routine clinical psychiatric practice. This paper describes the nature of the service which has evolved and discusses some of the major issues that were confronted in attempting to integrate medical and psychosocial interventions within a routine, psychiatric service.

Considerations in the development of routine services

The problem of engagement

Attempts to engage families in a programme of training and support does not always meet with success, which is surprising in view of the well documented burdens they face (Gibbons *et al*, 1984). Table II summarises the non-compliance rate for six family intervention studies including our own (in progress). For different reasons, figures for non-compliance in three of the studies are incomplete. Figures for the remaining studies range from 14% to 35%. The Hogarty *et al* (1986) intervention is the largest and most well controlled study to date, and it is likely that their figure gives the more accurate picture of the true non-compliance rate in the USA. The figure for our own sample, which is derived from a large urban inner-city area, is higher, and may possibly be due to the inclusion of low-EE families; all families were accepted into the study independent of EE status, and the therapists were blind to EE classification.

TABLE II
Compliance with family intervention

Study	% *refusing*	% *withdrawing*	% *non-compliance*
Hogarty *et al* (1986)	13	11	24
Leff *et al* (1982)	18	[1]	18+
Leff *et al* (1989)	4	26	29
Falloon *et al* (1985)	7	7	14
Goldstein & Kopeiken (1981)	[1]	8	8+
Tarrier *et al* (1988, and personal communication)	14	[2]	14+
Birchwood & Smith (ongoing)	21	14	35

1. Not reported.
2. All those withdrawing were included in the analyses.

From a service perspective, it is important to understand the factors contributing to non-compliance. We have identified three potential reasons which we have attempted to address in developing our service.

Firstly, some families feel threatened by the availability of a specialised service for them and construe offers of help as 'treatment' with the implication that the family is a 'problem' or has failed. As Kuipers & Bebbington (1985) indicate, offers of help might indeed reflect an assumption on the part of mental health professionals that relatives are in some way responsible for the development of the disorder, expressions of distress or anger by relatives serving only to confirm this view.

Secondly, a significant proportion of our families refusing help are relatives of first-admission patients: some emphatically deny the presence of a mental illness; some regard it as a single isolated episode and see no need for long-term advice and support, while in other cases, relatives' distress is translated into anger towards the professionals involved. Engaging first-admission families in a therapeutic programme requires due cognisance of the emotional impact that a diagnosis of schizophrenia can bring.

Finally, many families do not accept or understand the rationale of family intervention. In some instances, families make unrealistic demands that their relative should be 'cured', and do not see their role in this process or the relevance of components of the intervention; others withdraw because they perceive their needs as few or feel they have already been met (e.g. by education).

Serious attempts to develop routine services to families should confront the origins of the engagement problem. The service model described in this paper incorporates three crucial elements to address this issue. These are discussed in detail in the second half of this paper but include:

(a) attempts to foster an ethos of 'informed partnership', so that families and individuals feel they are actively involved in defining their needs and planning an intervention programme
(b) education and training of 'front-line' professionals to highlight the needs of families and to provide accurate information about schizophrenia
(c) developing interventions which are flexibly tailored to the needs of the individual and his family and involve rigorous outreach.

Clinical advantages and disadvantages of EE

High EE and relapse

It has been suggested (Leff *et al*, 1982, p.130), and often assumed, that high-EE families should be the priority focus of a family service. This has much to recommend it since high EE predicts certain kinds of behaviour (critical, overinvolved, rejecting) and is strongly associated with relapse. The adoption of high EE (fundamentally a research definition and not a diagnosis) as the primary index of need has limitations which must be carefully weighed when deciding *who* the recipients of family interventions are to be.

High EE as an index of service need

The high-EE family can be reliably identified only during an acute admission (Leff & Vaughn, 1985). The adoption of acute admission as an entry point limits the opportunity to engage individuals and families, and overlooks the significant number who are not regularly admitted (some of whom may logically include high-EE families). Indeed, it has been suggested that the needs of those with little contact with psychiatric services and their families may be equal to or greater than those with much more contact (Johnstone *et al*, 1984). Thus, the adoption of EE to assess need will overlook a significant number of individuals and families, notably those with low EE or less service contact.

High EE as a typology

The concept of the 'high-EE family' has acquired an unfortunate (though no doubt unintended) pejorative connotation of 'the problem family', which many families strongly object to, as they see it as a reification of the stereotype of the family as inherently pathological and uncaring (Hatfield, 1987). In spite of efforts to temper this view (Kuipers & Bebbington, 1985) and to present alternative 'educational/coping' models (Birchwood & Smith, 1987*b*), the impact of the research literature has, in the author's view, tended to potentiate a powerful negative stereotype which does not acknowledge the strength or capacity of the family for change and their support of the affected individual (Hatfield, 1987; Hatfield *et al*, 1987; Kantor *et al*, 1987).

High EE and social outcome

The poor quality of life which many individuals with schizophrenia can experience is a source of major concern to themselves and their families alike (Creer & Wing, 1973) but is a feature that intervention studies with high-EE families, with the strong emphasis on reducing risk of relapse, have tended either to overlook or to assign as a subordinate goal. It has not been demonstrated that high EE predicts poor social outcome or that low-EE families possess the necessary skills to promote social reintegration and maintain well-being. In fact, it is our experience (Birchwood & Smith, 1987*a*) that in some cases low-EE families have actively resorted to management strategies characterised by disengagement and distancing, which serve to promote social withdrawal and poor social adjustment, in an attempt to cope with an otherwise difficult and challenging family member, an observation also made by Hatfield *et al* (1987) and Vaughn (1986).

High EE and family well-being

The prime focus of family interventions has been the reducing of the risk of relapse. Relatively little attention has been paid to the needs of the family caring for that individual. Many families experience significant burden and stress in caring for a relative with schizophrenia and the illness can have a significant impact upon family life and emotional well-being, imposing social and personal hardships, straining family relationships and producing symptoms of stress (Gibbons *et al*, 1984). There is evidence that patient outcome, in terms of relapse and social adjustment, is related to family coping efficacy and style, loss of family cohesion, inconsistency in management decisions, and excessive family burden, which appear to predispose to a failure to cope (Birchwood, 1983). Broadening the focus of family intervention to help the family to cope by directly assessing the needs and well-being of the family may well indeed be crucial to the success of family intervention and long-term patient outcome (Falloon & Penderson, 1985; Falloon, 1988).

The temporal stability of EE

An underlying assumption is that EE represents a stable, enduring, trait-like quality. From the perspective of a service where the high-EE group is to be the primary focus, any temporal instability of high EE will mean that clinical time might be wasted on families who change from high to low EE or families may be overlooked where a change from low to high EE occurs. There is now growing evidence that a sizeable minority of high-EE relatives become less critical with the passage of time (Dulz & Hand, 1986; Hogarty *et al*, 1986; Tarrier *et al*, 1988) which is probably related to the quiescence of the patient's disturbed behaviour (Brown *et al*, 1972). Hogarty *et al* (1986), for example, report that 25% of their high-EE control group 'spontaneously' changed to low EE (v. 39% of those receiving family intervention). On the other hand, low-EE relatives tend to remain low, although Tarrier *et al* (1988) did report a minority who changed. Whether these changes reflect purely measurement error, a reversion to 'type', or a genuine fluidity in the characteristic is not yet known. Kuipers & Bebbington (1988) suggest, in effect, the second of these options in their subdivision of the low-EE group into 'good' and 'poor' copers, with the latter predicted to change in times of stress (e.g. hospital admission). An alternative perspective would eschew the concept of EE as reflecting an enduring trait and suggest that EE is an operational 'thermometer' of stress in a *particular* relative–sibling (or spouse) relationship at a *particular* time. In this sense, EE is characterised as a 'snapshot' of the state of ongoing complex transactions between the individual and the relative (Birchwood & Preston, 1990).

In summary, the constraints of high EE were felt to be too great to adopt it as the *primary* entry criterion to a service, although its use is clearly vital to inform practice if repeated relapse is a problem. Families should be given equal opportunity of access on a needs basis at a multiplicity of contact points, not just acute admission, a policy adopted in our service at All Saints' Hospital.

Importance of needs-led, goal-defined interventions

The goals of family interventions are not always made explicit, but a reduction from high to low EE is regarded as the principal indicator of the success of an intervention (i.e. manipulation of the independent variable). The means by which this is achieved varies widely, from structured problem-solving and communication skills training (Falloon *et al*, 1985) to the prompting of interactions between high-EE and low-EE relatives (Leff *et al*, 1982). This variability in content may be due to the lack of a clearly articulated model of the high-EE family interior to inform their design. The availability of goal-orientated steps in family intervention is important, as this provides the therapist with the means to gauge whether the intervention is proceeding appropriately and thus to enable any corrective action to be taken. More importantly, if the goals of family intervention are to be wider than that of relapse, to address family well-being and patient quality of life and respond to families according to need, then it is unclear how interventions focused on families of those with a high risk of relapse are to achieve this.

We have argued elsewhere (Birchwood & Smith, 1987*a*) that flexibility in stated goals of family interventions can best be achieved if they are structured to relate logically to the needs of the individual and the family in adjusting to the emergence of schizophrenia. In this way, interventions can be individualised and enable research to address the crucial question of what kind of intervention is appropriate for what kind of family, for what kind of outcome.

Service delivery issues

Family interventions undertaken in London (Leff *et al*, 1982), California (Falloon *et al*, 1985), and Pittsburgh (Hogarty *et al*, 1986) have all made demands on resources in terms of manpower and time (the latter for example requiring more than a year of close contact with the patient and family after discharge). These demands on resources must of course be offset against savings which accrue from fewer readmissions. If the benefits of family interventions are to be fully realised in clinical practice, then a broadly similar investment is required, at least initially; anything less surely confounds the logic of the enterprise. A mode of service delivery needs therefore to be established which is capable of delivering the intervention at a reasonable cost without prejudicing quality. Workers in the mental health field will be increasingly aware of 'grassroots' initiatives to meet the needs of families, as well as efforts by clinicians to incorporate advice provided by experts in family treatments into clinical practice (Kuipers & Bebbington, 1985). While these are important and must be encouraged, it must be borne in mind that there is as yet no evidence to suggest that these initiatives can mimic the effectiveness of the published interventions. Thus a service which aims to achieve the objectives promised by the published studies should assign quality assurance a high priority.

Community resources and rehabilitation policy

It is widely acknowledged that the improvements in community tenure which followed the introduction of neuroleptics in the early 1960s, increased instead of decreased the need for social rehabilitation and community care. There is no evidence to suggest that any further increase in tenure that might derive from family interventions will have a different effect (Hogarty *et al*, 1986). Notwithstanding this, family interventions are unlikely to prove to be a panacea for the community management and reintegration of the individual with schizophrenia, especially since the long-term impact of family intervention on the illness has yet to be determined (Hogarty *et al*, 1986). Families themselves have voiced disquiet about the increased burden which the 'community movement' is likely to place upon them: they fear that they might come to be regarded by managers simply as an inexpensive community resource (Hatfield, 1987). The prevailing ethos of community management demands that greater resources be directed towards supporting families of the severely mentally ill, but this in our view will only be effective if it is applied in the context of a rational, comprehensive system of case management, rehabilitation, and community services (Birchwood *et al*, 1988).

Implications for service provision

In summary, it has been argued that the availability of family interventions within a psychiatric service does not guarantee that help of the requisite quality reaches those in need. Thus we would suggest that an effective service should fulfil the following requirements:

(a) actively engage families, through a multiplicity of contact points
(b) be led by need
(c) ensure that family needs are understood and responded to sympathetically within a framework of partnership
(d) be integrated with existing treatment facilities
(e) provide ongoing training and supervision of 'front-line' professionals
(f) give a high priority to service quality.

A family service model

The 'Family Centre for Advice, Resources and Education' at All Saints' Hospital, was established in 1984 with the remit to develop and evaluate interventions for individuals with schizophrenia and their families within the context of a routine psychiatric service. In attempting to integrate family intervention with mainstream psychiatric treatment, careful consideration was given to the issues outlined in this paper. Below we sketch the service innovations which comprise the model currently undergoing evaluation to show how these considerations can inform clinical practice and service provision. A more detailed description of the implementation and evaluation of this model is beyond the scope of this paper but will be the subject of a forthcoming article (in preparation).

(a) Active engagement of families at multiple entry points

The service does not use high-EE status or acute admission only as an entry point. Instead, the centre routinely initiates contact with individuals and their families at multiple-entry points in the service including acute admission units, out-patient and maintenance medication clinics, day centres, and voluntary groups such as NSF and MIND. Service availability is not determined by referrals or specific individual requests for help but is offered routinely to families as one component of the psychiatric service.

(b) Needs-led interventions

Once an offer of help has been accepted, the specific characteristics and needs of the individual and family are assessed using various questionnaires and interview assessment measures. A needs/strength profile is derived from the information which is then used to plan a negotiated intervention. Services offered include:

(i) information and orientation to schizophrenia for patients and their families
(ii) helping the family and the individual cope and live with schizophrenia
(iii) training patients and families to recognise 'early-warning signs' of relapse
(iv) self-management of persisting symptoms.

The clinical aspects of these services are described in detail elsewhere (Birchwood, 1986; Birchwood & Smith, 1987*b*; Smith & Birchwood, 1987; Birchwood *et al*, 1989).

(c) Promotion of an ethos of 'informed partnership'

Efforts are made to ensure that family needs are understood and responded to sympathetically within a framework of partnership (Hatfield, 1979). The approach with families is one which acknowledges the difficulties that families may face in caring for a relative with schizophrenia and sees the family as facing problems rather than as a 'problem family', in need of treatment. The emphasis is on helping the family to develop the necessary expertise through the provision of information and skills for the family to determine the most appropriate management strategies for the problems they face, rather than prescriptively advising solutions which families then follow (cf. Falloon *et al*, 1985). A crucial component is the negotiation of the goals of the intervention with the individual and the family: this not only defines needs and goals but also serves to overcome the 'adversarial set' between professional and family which might be exacerbated when a 'therapy' for the family is offered.

(d) Integration with community agencies and psychiatric services

The family service has developed as a routine part of the total services offered to individuals with schizophrenia and their families. Close liaison is maintained with key professionals involved with a particular individual and through joint working and regular case reviews. Efforts are made to ensure that there is co-ordination and continuity in care and that the goals of family intervention are in line with the overall treatment and rehabilitation plans for a given individual.

The goal of maintaining patients in the community with their families requires the full use of community resources, both statutory and voluntary. The centre liaises closely with community agencies relevant to the particular needs of the individual and family. For example, in meeting the social and recreational needs for a given individual, use might be made of local community, recreation and adult education centres, social interest clubs or, if more specialised help is required, contact clubs and local day centres.

(e) Training and supervision of 'front-line' professionals

The availability of sufficient numbers of appropriately trained staff was identified as key factor affecting the efficacy and continuity of family interventions within a routine psychiatric service. A crucial function of the family service is therefore one of dissemination, training, and supervision. Three levels of training are routinely provided through in-service workshops and routine input to pre- and post-qualification training for all front-line professional staff:

(i) highlighting the needs of individuals with schizophrenia and their families
(ii) responding to the information and emotional needs of individuals and their families following a diagnosis of schizophrenia
(iii) helping families reduce the emotional and practical burden of schizophrenia

A central resource of materials and expertise is maintained at the centre but in addition trained 'tutors' are identified as supervisors to whom staff can relate directly for information, support, and supervision. Back-up information materials are provided in the form of a teaching pack with a training manual, information booklets (Smith & Birchwood, 1985), and videos. The specific interventions are then offered routinely to families from staff trained in their use on the admission units, out-patient clinics, and in various community settings. This training and supervision structure thus promotes the long-term continuity of family interventions within the psychiatric service.

(f) Quality assurance

All aspects of the service are closely monitored and evaluated to ensure that the objectives of the intervention are being fulfilled and that the quality of service provision is maintained. Efforts to ensure that the dissemination of skills to front-line professionals does not dilute the intervention or prejudice quality are encouraged through the continued maintenance of the family centre as a central resource, together with the established system of 'in-house' supervision and training described above.

Concluding comment

The results of psychosocial intervention studies over the past ten years has raised expectations among individuals with schizophrenia, their families, and, not least, psychiatric professionals. Translating these advances into clinical practice represents a major challenge for the psychiatric services in the years ahead and requires careful planning if the achievements and quality of these interventions are to be maintained and made available to those in need. We feel that the development of effective services, whether based in hospitals or in the community, needs to acknowledge and be influenced by the issues raised here in order to translate theory effectively into practice.

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The TAPS Project. I: Previous Psychiatric Diagnosis and Current Disability of Long-Stay Psychogeriatric Patients

A Pilot Study

JEREMY ANDERSON

Of all 89 long-stay residents in four psychogeriatric wards at a large London psychiatric hospital, 18 had a previous functional psychiatric diagnosis. Although the total group was markedly impaired overall, male sex, younger age, and a previous functional diagnosis were independently associated with better cognitive performance. Males and those with previous functional disorders also had less behavioural disability. These subgroups within the psychogeriatric in-patient population must be recognised if an in-built distortion in community reprovision of hospital psychogeriatric services is to be avoided.

Who are the long-stay psychogeriatric patients in our psychiatric hospitals? As the government's policy of psychiatric hospital closure develops, the limitations of current knowledge about this highly disabled group become apparent. From a whole-hospital survey Levene *et al* (1985) concluded that "the large existing number of elderly disabled patients . . . presents the most serious obstacle to hospital closure". Two-thirds of their subjects aged over 65 years had a primary diagnosis of dementia, but the types of disability associated specifically with this diagnosis were not reported. Diagnostic factors are important however. Kay (1962) showed differing survival rates among elderly patients in a psychiatric hospital suffering from 'functional', 'organic' or 'mixed' mental disorders. Range of disability is also important. It is known that levels of cognitive impairment (Whitehead & Hunt, 1982) and behavioural disability (Donaldson & Jagger, 1984) predict survival among the elderly. Unfortunately, studies of diagnosis and disability are rarely integrated, particularly with long-stay psychogeriatric subjects. This represents a gap in the information available for planning appropriate community reprovision of psychogeriatric services.

The present study had two aims: to explore the use of different assessment schedules with long-stay psychogeriatric patients; and to examine the relationship between diagnosis and disability in this group. To do this, patients' previous psychiatric diagnoses were correlated with their current disabilities.

The author is a member of the Team for Assessment of Psychiatric Services (TAPS), which is conducting a large-scale evaluation of the planned closures of Friern and Claybury Hospitals, in London. This particular study focused on the long-stay psychogeriatric in-patient services of the Islington District Health Authority, which are provided by four wards at Friern. These wards supply continuing supportive care rather than active rehabilitation. Since 1987 access to beds has been determined by a multidisciplinary 'bed bureau'. The admission criteria select patients with definite cognitive disabilities, whose behavioural problems (e.g. wandering, incontinence, fire-lighting) render care in alternative facilities impractical.

Method

The names of all in-patients resident on the four wards were obtained. Each patient was interviewed to assess cognitive function, and senior nursing staff were interviewed to provide behavioural information on each patient.

Cognitive functioning was assessed using the Abbreviated Mental Test Score (AMTS; Qureshi & Hodkinson, 1974) and the Mini-Mental State Examination (MMSE; Folstein *et al*, 1975). The original form of the MMSE was used, providing a maximum score of 30. The AMTS and MMSE questions were interleaved to reduce fatigue bias in the respondents. Reported behavioural functioning was assessed with the Modified Crichton Royal Behavioural Rating Scale (MCRBRS; Wilkin & Jolley, 1979), which rates patients on ten areas of problem behaviour, including items relating to both physical and mental care needs.

Each patient's age, sex, and date of current admission to hospital (not to present ward) were then recorded. The patient's age at first psychiatric contact was obtained from hospital records.

The diagnosis recorded in case notes at the time of first contact with psychiatric services was regarded as the 'original' diagnosis. Where differing diagnoses had been made subsequently, the most consistent was taken. Patients were allocated to either an 'organic' or 'mixed' group on the basis of their original diagnosis. The 'organic' group comprised those with a previous diagnosis of organic mental disorder only. Where both a previous diagnosis of functional psychiatric disorder and subsequent cognitive disability were specifically documented, the patient was assigned to the 'mixed' group. For the purposes of this classification paraphrenia was regarded as a functional disorder (Holden, 1987).

Because the disability data (AMTS, MMSE and MCRBRS scores) were not normally distributed, non-parametric summary statistics are used. Arcsine transformations were performed on these data before Student's t-tests were performed. The other data (age, first psychiatric contact, and length of current admission) are summarised parametrically and were not transformed before analysis. All statistics were calculated using SAS software (SAS Institute, 1985).

Results

Eighty-nine patients were identified on the four wards, but as one of these was subsequently found to have a history of an exclusively functional psychiatric illness (recurrent depression), with no record of cognitive disability on case-note review, he was excluded. The remainder comprised 18 'mixed' and 70 'organic' patients. For five patients the AMTS and MMSE were not completed, three because of severe perceptual disability and two because of difficulties communicating in English. Thus a total of 88 behavioural-problem assessments and 83 cognitive-disability assessments were analysed.

Table I shows the distribution of both previous psychiatric diagnoses and aggregated diagnostic group, by sex. There are proportionally more women than men in the 'organic' group (74% women, 26% men) compared with the 'mixed' group (61% women, 39% men), but this is not statistically significant (Fisher's exact test, two-tailed, $P=0.379$).

The women were significantly older than the men both at interview (82.3 ± 6.7 v. 75.2 ± 11.0 years, respectively, $t=2.97$, $P=0.005$), and at the time of their first psychiatric contact (74.8 ± 12.3 v. 66.8 ± 20.6 years, $t=2.17$, $P=0.03$). Length of current admission was not significantly different (3.8 ± 7.1 v. 4.2 ± 9.4 years). 'Organic' patients were older than 'mixed' patients when interviewed (81.3 ± 8.2 v. 76.3 ± 9.8 years, $t=2.14$, $P=0.03$), were older at initial psychiatric contact (77.5 ± 8.3 v. 52.9 ± 20.8 years, $t=7.68$, $P<0.0001$), and had shorter current admissions (2.3 ± 2.4 v. 10.2 ± 15.1 years, respectively, $t=4.19$, $P=0.0001$).

TABLE I

Sex and original psychiatric diagnosis

Psychiatric diagnosis[1]	*Men (n = 25)*	*Women (n = 63)*
'Organic' group		
senile dementia	14	48
multi-infarct dementia	4	4
total	18	52
'Mixed' group		
affective disorder	2	4
schizophrenia	4	2
paranoid states	1	4
mental handicap	0	1
total	7	11

[1] Original psychiatric diagnoses have been grouped by the following ICD-9 codes (World Health Organization, 1978): senile dementia 290.1–290.3, 290.9, 294.1; arteriosclerotic dementia 290.4; affective disorder 296, 300.4; schizophrenia 295; paranoid states 297; mental handicap 317.

The median scores for completed AMTS and MMSE assessments are summarised by sex and diagnostic group in Figs 1 and 2. Although the sample was severely cognitively impaired overall, there were consistent differences between groups. Both women and patients with a history of organic mental disorder alone were significantly more cognitively impaired than other patients. Few of the more disabled patients could answer any AMTS questions at all. While the scores from the less disabled were more evenly spread, with one male 'mixed' patient attaining a score of 8/10 on the AMTS for example, these still revealed significant disability. The MMSE results showed a greater range of scores than the AMTS, although these were still skewed towards high disability for all groups.

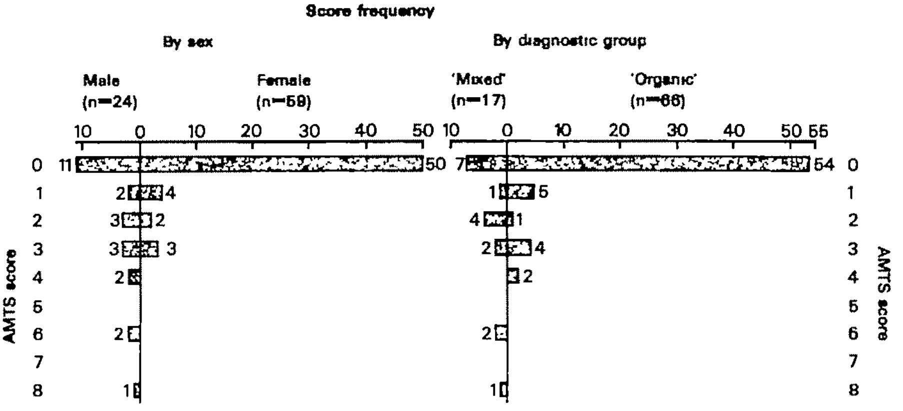

FIG. 1 AMTS cognitive score frequencies by sex ($t=4.68$, $P<0.0001$) and by diagnostic group ($t=4.25$, $P=0.0001$). Raw frequency scores are shown alongside respective columns. Low scores indicate severe disability. Maximum AMTS score = 10.

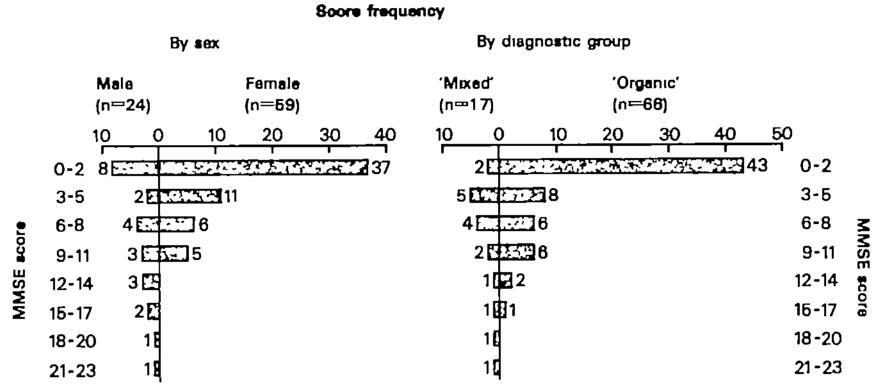

FIG. 2 MMSE cognitive score frequencies by sex ($T = 4.76$, $P < 0.0001$) and by diagnostic group ($t = 4.34$, $P < 0.0001$). Raw frequency scores are shown alongside respective columns. Low scores indicate severe disability. Maximum MMSE score = 30.

Total MCRBRS scores are summarised by sex and original diagnosis group in Fig. 3. These results recapitulate the scores for cognitive impairment: women and patients with a history of organic mental disorder alone had significantly more behavioural problems than other patients.

Because female patients were significantly older than males, and because of the possibility of an interaction between sex and previous psychiatric diagnosis, a three-way analysis of variance was performed. The effects of patient age, sex, and psychiatric diagnostic group were assessed against AMTS, MMSE, and MCRBRS scores (Table II).

TABLE II

Analysis of variance: sex, age and diagnostic group by AMTS, MMSE and MCRBRS scores

	AMTS		*MMSE*		*MCRBRS*	
	F	P	F	P	F	P
Age	5.71	(0.001)	3.38	(0.01)	0.63	(0.64)
Sex	8.12	(0.006)	9.14	(0.003)	5.61	(0.02)
Diagnostic group	8.74	(0.004)	6.38	(0.01)	7.19	(0.01)

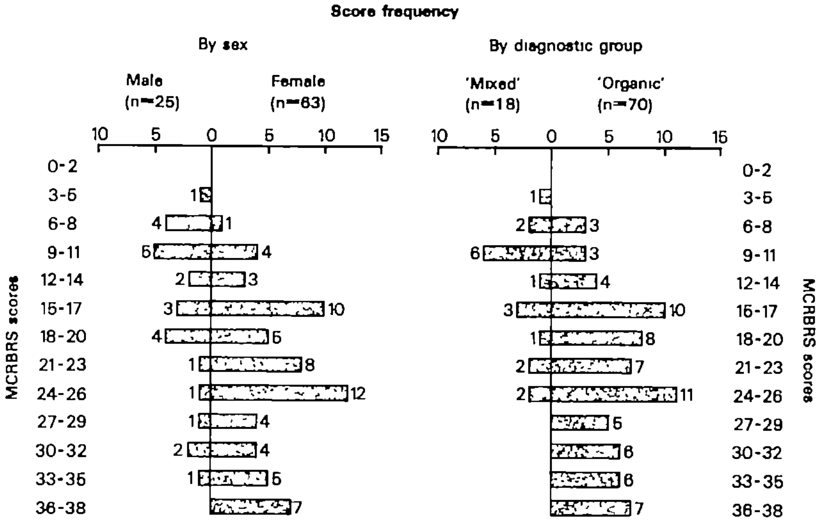

FIG. 3 MCRBRS behavioural score frequencies by sex ($t = 3.66$, $P = 0.0004$) and by diagnostic group ($t = 4.41$, $P < 0.0001$). Raw frequency scores are shown alongside respective columns. High scores indicate severe disability. Maximum MCRBRS score = 38.

With regard to cognitive impairment, patient age, sex, and diagnostic group each had a significant independent association with both AMTS and MMSE scores. For reported behavioural disability, patient sex and diagnostic group alone were independently associated with MCRBRS scores. There is no evidence of interaction between any of the independent variables with regard to AMTS, MMSE or MCRBRS scores.

Discussion

In this study of in-patients on long-stay psychogeriatric wards at Friern Hospital, over a fifth had a previous functional psychiatric diagnosis. These patients were younger and less disabled, cognitively and behaviourally, than the other patients. They were younger at original psychiatric contact and had longer current admissions than 'organic' patients.

What possible limitations are associated with these results? The study was subject to the same qualifications as others relying on case-note records for diagnostic assessment. For this reason patient disability assessments were performed before assignment to diagnostic groups. The simplest possible classification of previous psychiatric diagnosis was adopted to aid consistency. Although five instances of unclear previous diagnoses were found, a consensus diagnosis was apparent in each case, and none of these affected assignment to the aggregated diagnostic groups. While the composition of psychogeriatric wards could vary over time, and between institutions, no factors could be identified which would make the studied wards atypical of psychogeriatric wards in large psychiatric hospitals throughout the UK. Absolute similarity to other groups of psychogeriatric in-patients with regard to cognitive disability cannot be accurately assessed since directly comparable studies do not exist. However, the pattern of reported behavioural disability (MCRBRS score) of these subjects is similar to that of the psychogeriatric in-patient subgroup described by Charlesworth & Wilkin (1982). This does suggest that these findings can be generalised.

With regard to the first aim of the study, the methodological difficulties of assessing this group of severely disabled patients have been clearly illustrated. Both AMTS (Qureshi & Hodkinson, 1974) and MMSE (Folstein *et al*, 1975) scores have been used in the past to discriminate between patients with organic and functional psychiatric illnesses. There is additional support for this use of the AMTS since it is derived from the Roth–Hopkins test, which correlates well with pathological brain changes at autopsy (Blessed *et al*, 1968). However, the use of neither test has been reported with patients having 'mixed' psychopathology, and their use with severely disabled long-stay in-patients is limited. The previously derived discriminatory cut-off points cannot be assumed in this instance. Thus no conclusions about current psychiatric diagnosis can be drawn from the test performances of the 'mixed' group. It is of interest that the MMSE provided a broader range of scores than the AMTS, even in the most disabled groups, presumably by testing a broader range of cognitive functions (Roth *et al*, 1986). The MMSE seems the preferable cognitive test, although recent work concerning the influence of respondents' demographic background upon test scores should be noted (Jorm *et al*, 1988).

The MCRBRS showed a wider range of scores, and also discriminated well between subgroups. In contrast to the cognitive scores, behavioural ratings were not independently associated with age when analysis of variance was performed. This is in accord with Charlesworth & Wilkin's (1982) observations using the MCRBRS with psychogeriatric in-patients. The difference between cognitive and behavioural disability scores with respect to age strengthens the assumption that these are different aspects of patient performance.

The major finding of the present study is that these long-stay in-patients can be classified into subgroups of differing cognitive and behavioural disability on the basis of both sex and previous psychiatric diagnosis. That elderly mentally ill women in long-term care are more disabled than men has been reported by Willcocks *et al* (1987), and is not discussed further here. The significance of a previous functional psychiatric diagnosis in relation to decreased cognitive and behavioural disability among psychogeriatric in-patients has not been noted previously.

A past functional psychiatric diagnosis has social implications which may explain this finding. Chronic psychiatric patients may have less social support than those developing dementia from a state of good mental health. Low social support is known to increase admission to psychogeriatric wards (Bergmann *et al*, 1978). Also, the resistance to psychogeriatric care known to exist among the general population (Willcocks *et al*, 1982) may be diminished among institutionalised chronic psychiatric patients. Concerning staff, it is known that doctors, both general practitioners and psychiatrists, act as 'filters' (Goldberg & Huxley, 1980) in the flow of patients into specialist care. Perhaps these filters operate differently when patients have had previous contact with psychiatric services, easing access to psychogeriatric care.

From a biological viewpoint, the findings of the present study are made difficult to interpret by gathering a diverse range of diagnoses into aggregated groups. For example, some functional disorders, like schizophrenia (Johnstone *et al*, 1981; Cutting *et al*, 1983) and paraphrenia (Naguib & Levy, 1987; Levy *et al*, 1987), have been associated with cognitive deterioration. This might suggest an accelerated requirement for psychogeriatric care among these patients, but these interactions cannot be explored here because of the heterogeneity and small numbers in the 'mixed' group.

The significance of the present findings for the community reprovision of psychogeriatric in-patient services is in two areas. First, in immediate planning terms, it appears that relocation of long-stay hospital beds for the elderly mentally ill will be predominantly to smaller long-stay facilities elsewhere, although with a more varied range of management styles and services (Blessed, 1988). If disability is the major criterion in relocation, without reference to past psychiatric diagnosis, a clustering of patients will occur. For example, male patients with a previous functional psychiatric diagnosis will be over-represented in the facilities with least supervision. Whether or not this clustering is clinically desirable, which is an open question, it has implications for service provision and staffing that have not been previously recognised.

Second, there are possible long-term implications. The shape of community psychiatric services will be strongly influenced by service reprovision at the time of hospital closure. There is a danger that the future structure of community care will reflect the needs of the current hospital in-patients rather than those of the community population. The ratio of one in five for long-stay psychogeriatric patients with a previous functional psychiatric diagnosis is unlikely to be replicated among those in the community requiring long-stay psychogeriatric care in the future. This raises the prospect of an in-built distortion in community service reprovision before it has even begun, by underestimating the requirement for high-dependency beds.

This pilot study raises several further questions. Can these findings be replicated with more sophisticated forms of diagnostic assessment? Do patients with comparable 'mixed' psychopathology exist in other forms of psychiatric care, for example rehabilitation wards? If so, will investigation of these patients clarify the decision-making process by which the selection of type of in-patient care is made? Is there evidence of different degrees of cognitive disability among elderly psychiatric hospital in-patients according to their previous psychiatric diagnosis? These questions will be addressed in a future study.

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Depression among Elderly Residents of Local-Authority Residential Homes
Its Nature and the Efficacy of Intervention

DAVID AMES

Of 390 residents in 12 local-authority homes for the elderly, 93 had evidence of depression on screening and underwent standard clinical assessment. Half had an affective disorder, and a further third had depressive symptoms in the setting of an organic mental disorder. The 93 residents had a high rate of physical illness and disability, undertook little social activity, and were visited infrequently. A range of interventions for depressed residents, the majority social in type, mostly proved difficult to implement. There was no evidence of efficacy of psychiatric intervention at three months; at one-year follow-up a quarter of study participants had died, and 28% of those who were reassessed for depression showed evidence of recovery.

About 5% of British people aged 65 and over live in some form of institutional care, and the largest proportion are cared for in local-authority residential 'Part-III' homes (Day & Klein, 1987). Although these homes were established to cater for those elderly not suffering from illness (Ministry of Health, 1948), evidence that most residents of such homes are physically or psychiatrically ill has accumulated (Carstairs & Morrison, 1971; Lowther & McLeod, 1974; Wilkin *et al*, 1978; Gurland *et al*, 1979; Mann *et al*, 1984*a*).

Dementia has a high prevalence among residents of Part-III homes (Lowther & McLeod, 1974; Mann *et al*, 1984*a*), and appears to be becoming more common in this population (Ames *et al*, 1988). In addition, many elderly residents are depressed. Of 200 elderly individuals admitted to a large Part-III home in Edinburgh, 9% had depression diagnosed by a psychiatrist, and a further 40.5% had dementia (Lowther & McLeod, 1974). From the analysis of a standard interview, 38% of residents without severe dementia living in 12 Part-III homes in Camden, London, showed 'clinically important' symptoms of depression (Mann *et al*, 1984*a*). The prevalence of depression was similar (34%) in these 12 homes three to four years later (Ames *et al*, 1988). Overseas studies have also reported high rates of depression among elderly people in institutional care (Teeter *et al*, 1976; Snowdon, 1986; Spagnoli *et al*, 1986; Lau-Ting *et al*, 1987).

One problem in drawing inferences from epidemiological research on depression rates is the variety of definitions employed by investigators. A clear distinction can be made between studies reporting depressive illness and those reporting symptoms alone (Swartz & Blazer, 1986). In discussing the high prevalence of depressive symptoms in Camden's Part-III homes (Mann *et al*, 1984*a*), Pitt (1986) commented "What is one to make of such a finding? Are depressed old people selectively admitted to old people's homes. . .? Or did the survey turn up a number of apathetic querulous self-absorbed old people feeling stranded and somewhat stigmatised, with too much time to brood and too little else to do?" The next step in research would seem to be to define the type of disorders detected by screening for depression in Part-III homes.

Excellent evidence exists for the efficacy of physical treatments in depression of severe and moderate degree at all ages (e.g. Medical Research Council, 1965; Kendell, 1981). However, evidence that the milder depression seen in older subjects living in the community can be treated effectively is lacking (Baldwin & Jolley, 1986). There is no published study reporting attempts to treat depressed elderly residents of Part-III homes. In recent years the standard dictum that affective disorders among the elderly have a good prognosis has been called into question (Murphy, 1983; Magni *et al*, 1988). Follow-up of 110 residents of Part-III homes classed as depressed by a brief interview showed that after three to four years only 35 remained in their original institution, most who had left were likely to be dead or in hospital, and only six survivors had recovered from depression (Ames *et al*, 1988). So poor an outlook suggests an urgent need to determine what factors might improve the prognosis and lower the prevalence rate.

The current study was to answer three questions. From what types of depression do residents suffer? Can a psychiatrist visiting homes on a regular basis lessen the impact of depression by using available resources over three months? What will be the outcome for depressed residents one year after detection, and what factors will influence their prognosis?

Method

Screening

The details of the screening procedure have been described elsewhere (Ames *et al*, 1988). In summary, the residents of the 12 Part-III homes managed by the London borough of Camden were screened with the Brief Assessment Scale (BAS), a standard, valid instrument for the detection of depression and dementia (MacDonald *et al*, 1982; Mann *et al*, 1984*a*, 1989), derived from the Comprehensive Assessment and Referral Evaluation Schedule (CARE; Gurland *et al*, 1977). The BAS consists of an organic brain syndrome scale (OBS) and a depression scale (DEP). According to their score on the OBS scale, respondents can be classed as probably severely demented (score of 8), probably mild–moderately demented (score of 3–7), or probably not demented (score of 0–2). Respondents scoring 8 on the OBS scale have their interviews discontinued, but those who score 7 or less usually complete the DEP scale. A score of 7 or greater on the DEP scale (scored 0–24) has been shown to predict significantly depressed states, as judged by a psychiatrist experienced in work with the elderly (Mann *et al*, 1984*a*).

Screening by trained interviewers took place sequentially in the 12 homes, between July 1985 and May 1986. Residents who met study criteria for significant depressive symptoms without severe dementia (i.e. OBS ≤, DEP ≥7) were asked to participate in the current study.

Assessment

All residents who fulfilled the criteria for depression without severe dementia were assessed by the author with the following measures. The Geriatric Mental State Schedule (GMS; Copeland *et al*, 1976) was used to assess psychiatric symptoms. It is a standard, valid instrument for use in old age, which yields data suitable for diagnosis by the AGECAT computer program (Copeland *et al*, 1986). In order to assess the author's reliability in its use, ten study subjects were interviewed and jointly rated by the author and another experienced rater. The 17-item Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960, 1967) was used to grade severity of depression.

For assessing activities of daily living (ADL) a modified version (Mann *et al*, 1984*b*) of the method described by Katz & Akpom (1976) was employed. This schedule was completed with the head of home or deputy as respondent, and covered capacity for self-care with a scoring range of 0 (entirely independent) to 41 (totally dependent). In addition, the head of home was asked to rate each subject as not depressed, or mildly moderately or severely depressed, and how many visits the resident had had in the past month. All social activities undertaken in the previous week were recorded on a specially designed form. Each day was divided into five sections, so that the resident's activity (outings, group activities, being visited, etc.) could be coded for each session. A possible score of between 0 and 35 sessions occupied for the preceding week could be obtained.

The medical history of each resident was gathered from current medical records, prescription sheets, previous hospital records, and from the residents themselves.

Diagnosis

After assessment, data from the GMS, together with available historical data, were used to make a psychiatric diagnosis by DSM–III criteria (American Psychiatric Association, 1980). Subsequently the GMS data were processed by AGECAT to yield standardised diagnoses of eight psychiatric syndromes: organic, schizophrenic, manic, or depressive (psychotic or neurotic) illnesses, hypochondriacal, obsessional, phobic, and anxiety neuroses. The AGECAT output indicated which subjects fulfilled criteria for classification as 'diagnostic cases' (level 3–5) or 'subcases' (level 1–2) of disorder.

Interventions

When a resident had been assessed, the author, a consultant psychiatrist for the elderly, and a social worker with extensive experience of elderly psychiatric patients formulated interventions, intended to relieve depression, which would have been suggested had the resident been referred to the psychogeriatric team. Half the depressed residents (intervention group: IG) were offered immediate implementation of interventions; half served as a comparison group, for whom interventions were proposed and recorded, but not implemented in the initial three months of the study. As there was a possibility that residents in homes where interventions were taking place might derive some non-specific effect from the activities of the author within the home, it was decided to have two comparison groups: one in homes where intervention was taking place (comparison group 1: CG1), and the second (CG2) in three homes which were not visited by the author after initial assessment, until reassessment took place at three months. In the remaining nine homes, two-thirds of the depressed residents were randomly allocated to IG after controlling for sex of resident. The rest were allocated to CG1.

Interventions included advice to visiting general practitioners (GPs), making suggestions to senior care staff, and direct contact with relevant agencies. IG subjects were visited throughout the intervention period.

Follow-up

After three months of intervention, all participating residents were traced and when possible reinterviewed with the BAS by members of the original interview team, blind to psychiatric diagnosis and intervention status of residents. The HRSD and ADL ratings also were repeated. Residents in CG1 and CG2 were similarly reassessed, three months after initial assessment. Nine months later residents again were traced and similarly reassessed. Date and cause of death were obtained from death certificates of residents who died during follow-up.

Statistical analysis

The three groups (CG1 and CG2 both separately and combined) were compared at 3 and 12 months in terms of categorical differences in outcome (defined as scores <7 (no longer depressed) or $\geqslant 7$ (still depressed) on the BAS DEP scale) using χ^2 tests. Mann–Whitney *U* tests were used to assess differences between groups in changes of DEP, HRSD and ADL scores from baseline after 3 and 12 months. Correlations are expressed as Pearson coefficients.

Results

The results of the screening have been reported in detail (Ames *et al*, 1988). In summary, 390 of the 439 residents could be interviewed with the BAS, and 271 completed the DEP scale. Ninety-two residents fulfilled the study criteria for depression without severe dementia, and one resident who could not complete the DEP scale but who scored above 7 on a very limited interview also was included. The 93 participants comprised 17 men, 76 women, median age 82.3 years (range 65–107), median length of stay in home 2.4 years (range 0–20 years). Fifty-eight were classed as suffering from probable mild–moderate dementia by the OBS scale and 35 had no evidence of dementia detected at screening.

Three residents died before the GMS interview and four refused it. The median time between screening and GMS interview for 86 residents was 15 days. Eighty-eight residents could be rated with the HRSD. Recent social activity could be rated for 89, ADL scores, frequency of visitors, depression rating by the head of the home, and medication data were obtained for 91, and some information was gathered from hospital records or GPs' notes for all 93 residents.

Psychiatric diagnoses

The test of the author's reliability in using the GMS gave a mean weighted kappa (Cohen, 1968) for the 184 items comprising the 21 subscales of the GMS described by Gurland *et al* (1976) of 0.89.

AGECAT diagnoses were derived for the 86 residents who completed the GMS interview (Table I). Sufficient data were obtained to make diagnoses using DSM–III criteria for 92 residents (Table I). Whichever diagnostic system was used, about half the residents studied met specific criteria for a 'case' of depression, and a third of residents had an organic psychiatric condition. There was good agreement between the two systems, with only 12 discrepancies: four due to AGECAT's inability to diagnose schizophrenia or related disorders in residents where knowledge of past history permitted diagnosis under the DSM–III system; four where very old, ill, or foreign-born residents were diagnosed as organic cases by AGECAT but as cases of depressive disorder by DSM–III; and four where residents were classed as having mental retardation, mild primary degenerative dementia, atypical depression and an adjustment reaction by DSM–III, and by AGECAT as depressive neurosis level 3 (2) or subcases of disorder (2).

TABLE I

Diagnoses of 93 residents assessed as having depressive symptoms without severe dementia by the BAS

Diagnosis	*No. of residents*
AGECAT diagnoses[1]	
Depressions	*47*
subcases, level 1 or 2	6
depressive neurosis cases, levels 3 or 4	25
depressive psychosis cases, levels 3, 4 or 5	16
Organic disorders	*35*
subcases, level 1 or 2	7
cases, levels 3, 4 or 5	28
Schizophrenia	*2*
subcase, level 2	1
case, level 5	1
Anxiety neurosis	*2*
subcases, level 2	2
DSM–III diagnoses[2]	
Depressive disorders	*45*
major depressive disorder	25[3]
recent major depressive episode in partial remission	1
atypical depressive disorder	9
dysthymic disorder	8
adjustment disorder with depressed mood	2
Organic disorders	*29*
primary degenerative dementia, senile onset	23
multi-infarct dementia	1
dementia associated with alcoholism	1
dementia (following cardiac arrest, cerebral hypoxia)	1
alcohol amnestic disorder	1
mild mental retardation	1
alcohol dependence, continuous	1
Other psychiatric disorders	*7*
schizophrenic disorders	5
atypical paranoid disorder	1
dependent personality disorder	1
No DSM–III disorder detected	*11*
Insufficient data for even tentative diagnosis	*1*

1. For 86 residents completing the GMS.
2. For all 93 depressed residents.
3. Included one bipolar disorder in depressed phase.

Because the DSM–III diagnoses seemed to have greater clinical relevance, they are used in the remaining analyses.

Standard scale scores and factors associated with depression

The 88 study residents assessed were not a severely depressed group as judged by HRSD scores (median score 13, range 0–26). Scores were highest for 23 residents with a DSM–III diagnosis of major depressive disorder (median 19, range 14–26), and lowest for 11 with no current DSM–III disorder (median 6, range 0–9).

The DEP scores for the 93 residents ranged from 7 to 19 (median 10). The highest scores were recorded by the

TABLE II

Interventions proposed by psychogeriatric team for 90 residents participating in intervention study

Intervention	*No. of residents*
'Psychiatric' interventions	
Start/increase/review/cease psychotropic drug(s)	23
Review by previous treatment team	12
Involve community psychiatric nurse	6
Further assessment or management by psychogeriatrician	5
Admit to hospital under section 2 of Mental Health Act	1
Voluntary admission or day hospital	1
Other psychiatric intervention/advice	2
'Medical' interventions	
Refer to specialist service	27
Replace/obtain aids (glasses, hearing aid, teeth, etc.)	7
Start/increase/review/cease non-psychotropic drug(s)	4
Other medical intervention (e.g. syringe ears)	6
'Social' interventions	
Increase/involve in available activities	43
Move or review placement	12
Regular visitor	10
Other social intervention or assistance	14

25 residents with major depressive disorder (median 12, range 7–19), the lowest by 11 with no current DSM–III disorder (median 8, range 7–11).

The head of home rated 35 residents as 'not depressed', 33 as mildly, 16 as moderately, and 7 as severely depressed. The group rated as not depressed included 3 of the 11 residents without current DSM–III disorder and 13 with dementia, but it also included three with major depressive disorder. The severe group included six with major depression and one with an atypical depressive disorder.

The BAS OBS scores were highest among the 26 residents who had DSM–III diagnoses of dementia (median 6, range 4–7). No resident with an AGECAT 'organic case' diagnosis or a DSM–III dementia diagnosis had an OBS score below 4. Of the 58 residents with OBS scores above the previously validated cut-off point (2/3) for the detection of probable mild–moderate dementia, 26 had a DSM–III dementia diagnosis, 2 another organic condition, 11 major depression, 11 another affective disorder, 3 schizophrenia, and 5 had no DSM–III disorder detected by clinical assessment.

The ADL scores indicated some impairment for most of the 91 residents rated (median 9, range 0–33). Social activity levels were low and residents were visited infrequently. The median number of sessions occupied with social activity was 2 (range 0–15) out of the 35 per week for 89 residents. Of 91 residents for whom data were available, 39 were never visited at all, and 12 others less than four times per month.

It had been anticipated that the ADL scores would have a direct correlation with DEP and HRSD scores, and that these two depression ratings would have an inverse correlation with the number of social activities and the number of monthly visitors. No significant correlation between ADL and HRSD scores ($r=0.16$), or between ADL and DEP scores ($r=-0.10$) was found. Social activity ratings had no significant correlation with DEP scores ($r=-0.04$), or HRSD scores ($r=0.23$). Number of visitors received had no correlation with DEP ($r=-0.09$) or HRSD ($r=-0.19$) scores.

Historical data and information from case notes

Thirty-six residents had records of previous psychiatric in-patient admission, while a further 14 had been seen as out-patients or on domiciliary consultations by psychiatrists in the past. Only 9 of the 93 residents were in current contact with psychiatric services when assessed.

A quarter of the sample (23 residents, predominantly with depressive diagnoses) were taking antidepressants, and 24 residents were taking hypnotics. Seventeen were taking neuroleptic drugs, but only 4 had a diagnosis of schizophrenia, and only three others had any history of delusions or hallucinations.

Only nine had no physical condition which impaired the ability to cope independently. Forty subjects had three or more body systems affected. The commonest disorders were severe visual impairment (virtually blind) in 38%, muskuloskeletal disorders (37%), cardiovascular disorders (30%), and deafness (29%). Residents with a DSM–III diagnosis of major depressive disorder tended to have more physical disabilities than other residents.

Intervention study

There were 173 interventions proposed for 90 residents (three died before interventions were proposed). For six residents four interventions were suggested, for 20 three interventions, for 29 two, for 31 one, and for 4 residents no additional intervention was thought justified. The recommended interventions are listed in Table II.

Forty-four residents were allocated to IG, 22 to CG1 and 24 to CG2. The three groups were similar in age and DEP and OBS scores, although members of CG2 were more likely to be male (30%) than members of IG (11%) and CG1 (9%), and the comparison subjects had a longer median length of stay in Part-III accommodation (32.5 months for CG1, 40.5 months for CG2) than IG subjects (20.5 months).

Although 81 interventions were recommended for the 44 IG residents, only 27 of these could be implemented within three months, and half the IG residents did not have any recommended intervention implemented in that period. Two interventions recommended for CG1 residents were implemented by coincidence in the three months. Fifty-four interventions were not implemented for IG residents because of:

(a) activities not being increased for any resident in any group (18)
(b) failure of GPs, care staff or other agencies to respond to written advice within three months (19)
(c) refusal of a resident to accept offered help (12)
(d) resident dying before help could be organised (3)
(e) unavailability of recommended alternative accommodation (2).

Those interventions which were successfully implemented included 8 of 14 recommended prescription alterations, 8 of 14 recommended referrals, all 4 recommended follow-up actions by the psychiatric service, and 2 special investigations.

Three-month follow-up

After three months, six of the 90 residents who commenced the intervention study were dead, and five either refused further interview or could not complete the DEP scale. The outcome data (Table III) showed no significant differences between groups. The results from the two comparison groups were similar and are combined for display. The differences in recovery rates and DEP scores between groups were not statistically significant. A comparison of the 21 surviving IG residents who actually received some intervention, with the 20 survivors from that group who did not have any intervention, also showed no statistically significant difference in recovery rates. The HRSD and DEP scores at three months were positively correlated with one another ($r = 0.69$) and with the change in score from baseline ($r = 0.37$).

Twelve-month follow-up

Of the original 93 residents, 22 had died and one was too ill to be interviewed. Three residents refused further interview. Four residents had left their original Part-III home by the time of the second follow-up interview, but all were visited. One was in a family home under an adult fostering scheme, one was in a long-stay geriatric ward, one had moved to a voluntarily run nursing home, and one was in a different Part-III home. In all, 67 residents underwent BAS interview. Seven now had an OBS score of 8, and 60 were able to complete the DEP scale. The HRSD was completed for all 60 residents who completed the DEP scale. ADL data were obtained for 70 of 71 survivors. All scores were compared with data obtained at initial assessment, 12 months earlier (Table III).

Only nine further recommended interventions which had not been implemented in the initial three months were executed in the subsequent nine months. Categorical outcomes assessed by the DEP scale showed that 17 of 60 residents had DEP scores below 7, but there was no statistically significant difference between IG and CG residents in regard to the numbers 'recovered'. Twenty of the survivors who completed the DEP scale had had some recommended intervention implemented, 40 had not. Again the actual receipt of a recommended intervention did not render a resident more likely to recover from depression as defined by the BAS. Six of the 20 residents who had received an intervention recovered but so did 11 of the 40 who had not.

There was a high correlation between DEP and HRSD scores at 12 months ($r = 0.75$), and changes in scores obtained with the two instruments since initial assessment also were positively correlated ($r = 0.48$). The tendency for some fall in depression scores to occur, first noted after three months, was maintained, but there was no significant difference between intervention groups in the size of fall. ADL scores rose for all diagnostic groups.

A comparison of the 43 residents with DEP scores of 7 or greater with the 17 residents with scores below 7 showed

TABLE III
Follow-up for 90 residents who commenced intervention study

Variable	*Three-month results*		*Twelve-month results*	
	Intervention group	*Comparison groups*	*Intervention group*	*Comparison groups*
No.	44	46	44	46
Dead	3	3	10	9
Incomplete data	0	5[1]	4[2]	7[2]
DEP score <7 (recovered from depression as defined at screening)	11/41 (27%)	6/38 (16%)	11/30 (37%)	6/30 (20%)
Median change in DEP score since screened	−1	−1.5	−2.5	−2
Median change in HRSD score since assessed	−1	−1	−4	−2
Median change in ADL score since assessed	+1	0	+2	+2

1. Two refused, three too confused for DEP scale or HRSD.
2. Three refused, one too ill, seven too confused.

that the latter had spent less time in Part-III homes before the study, were more likely to have had a past psychiatric in-patient admission, and to have had lower initial depression scale scores. The group of 'recovered' residents included five with an initial diagnosis of major depressive disorder, of whom three were now in full clinical remission and two were markedly improved. Twelve other residents with major depression had survived but not recovered. Multi-infarct dementia had developed in two residents who had an initial DSM-III diagnosis of affective disorder.

The 22 residents who died (3 before the intervention study, 19 in the ensuing year) were likely to have been older, more impaired in their activities of daily living, and to have had more physical illnesses and disabilities when first assessed than the 71 who survived. Initial scale scores, intervention status, and psychiatric diagnoses were not good predictors of mortality. The causes of death recorded on the death certificates were chest infection (8), cardiac disease (6), cerebrovascular accident (2), cancer (1), pulmonary embolism (1), old age (1), cholangitis (1), septicaemia (1), bleeding peptic ulcer (1).

Discussion

This study provides new data concerning the relationship of the DEP scale and head-of-home assessment to the HRSD and GMS. The DEP scale correlated with the HRSD as a measure of severity and as a measure of change. The DEP scale originally was validated as a categorical measure, but the data suggest that severity of depression correlates with total score. The finding that the highest scores were in subjects with major depressive disorder, and the lowest ones in residents with no DSM-III diagnosis, supports the validity of the DEP scale. Heads of home appeared to have a higher threshold than the DEP scale for classing a resident as depressed, but some of these ratings were idiosyncratic. This is important, because care staff usually inform visiting doctors of residents' medical problems. There also was inconsistency in these ratings over time, as the head of homes sometimes changed.

This study also gave a chance to compare two diagnostic systems. Although AGECAT relies solely on cross-sectional data to make a diagnosis, there was impressive agreement with DSM-III diagnoses, supporting its validity. Most disagreements could be explained on the basis of historical information unavailable to AGECAT and a somewhat lower diagnostic threshold for AGECAT as compared with DSM-III.

The diagnostic results have confirmed the initial report (Mann *et al*, 1984*a*) that the DEP scale as a screen selects not only residents with depressive illness, but several with isolated depressive symptoms which are often part of another psychiatric condition (especially dementia), or associated with physical disability. However, the majority of residents so detailed were suffering from a disorder which merited the main diagnosis of a depressive syndrome: 55% of those classified by GMS/AGECAT, 49% of those who received a DSM-III diagnosis. The severity of these disorders was variable. Twenty-five residents had a major depressive disorder, others suffered from milder depressions, nearly all of which were chronic. The latter, often having been treated in hospital, fitted Post's (1972) description of 'depressive invalids' or 'continuous illness'. With falling numbers of long-stay psychiatric beds, and rising numbers of elderly in the population, it should be expected that more elderly people with chronic affective illness will be placed in Part-III homes.

About a third of residents who were classed as depressed at screening had dementia. The delay between screening and assessment was helpful in preventing misclassification of one or two residents who may have had an acute brain syndrome when screened. The data confirm an earlier report (Ames *et al*, 1988) that subjects classed by the BAS as both depressed and demented are likely to consist of a demented group with depressive symptoms, and a group who perform badly on the OBS scale because they are depressed. The five schizophrenics also identified serve to underline the fact that depression can occur in this condition (Hirsch, 1982). The 11 residents with no DSM-III disorder were very old or disabled. The problems of differentiating physical disorder from depression are well documented (Eastwood & Corbin, 1986).

Two-thirds ofthe DSM-III affective disorders and all the cases of schizophrenia were chronic. Although over half the residents had previous contact with psychiatric services, and over a third had been placed in the Part-III home directly from psychiatric wards, only 10% were in current contact with such services at the time of the study. Three possible explanations for this are:

(a) patients on wards run by psychiatrists not specialising in the care of the elderly are often discharged to Part-III accommodation; although psychiatric advice to these homes can be provided by the local psychogeriatric service there may at times be a failure to transfer the care of these discharged patients to that service

(b) psychogeriatric services do not routinely see all residents themselves, but rely on GPs to refer them

(c) many of the residents in this sample suffered from dementia, or chronic affective or schizophrenic disorders; the untreatable nature of the underlying disorder or the apparent failure of previous attempts at treatment may have discouraged further referral.

The depressed residents were inactive and infrequently visited. No evidence of any inverse association between degree of depression and activity levels or frequency of visits could be found, perhaps because these scores were so low. The low activity levels are consistent with earlier work showing that residents of Part-III homes are likely to spend two-thirds of the day doing absolutely nothing (Godlove *et al*, 1982). The high prevalence of physical disability and illness accord with findings from the previous survey of residents in Camden Part-III homes (Mann *et al*, 1984*a*). The association of depression and physical illness seems to be particularly strong in these homes, perhaps reflecting reasons for admission as well as partly explaining the poor prognosis of the depressive disorders in this population.

Intervention study

The intervention study was designed to test the proposition that one psychiatrist working in homes, using available services, could substantially reduce levels of depression. The interventions were not, in the main, 'psychiatric' in nature: they included recommendations of an increase in available activity for half the sample (outings, group discussions, music sessions, structured social interaction) and the provision of a regular visitor for ten residents.

The large number of 'medical' interventions suggested reflected the high prevalence of physical disability and illness, combined with a perception that such conditions were not being optimally managed in all cases. The low number of subjects for whom the prescription of antidepressants was advocated may reflect the biases of the psychogeriatric team. However, less than a third of depressed residents had major depressive disorder, which itself represents a rather broad definition of depression, and probably encompasses some conditions which are unlikely to respond to specific physical therapies. Another reason for any apparent therapeutic nihilism was the fact that several residents had had many psychiatric admissions and had been subjected to most available treatments for depression with little apparent benefit. Twenty-three residents had been prescribed antidepressant treatment already.

The implementation of recommended interventions was not easy. Increases in activity as advocated would have involved a modification of care practices and home environment and could not be implemented. Interventions that could be implemented usually took time to initiate. The arrangement of a visitor from a voluntary group was one offer frequently declined by residents. Co-operation from GPs in the alteration of prescribed medications and the referral of residents to specialist services was variable. Only about a third of interventions could be implemented within three months. Their implementation would have been easier if psychogeriatric services had a designated role in the management of homes and the medical care of residents.

The main instrument chosen to assess recovery was that which had detected depression in the first instance: the DEP scale. Interviewers were blind to treatment and initial diagnosis and thus rater bias was minimised. No significant outcome differences between groups were noted, and in particular there were no differences between IG residents who did or did not receive recommended interventions. Given the small number of interventions actually implemented, it still remains an open question whether the actions recommended by a psychogeriatric team would be effective.

Outcome at 12 months

Of surviving depressed residents interviewed at one year, 28% had improved on the DEP. It appeared that the prognosis of a depressive illness in a resident of a Part-III home was poor. Recovery from depressed mood, as defined by the DEP scale, was more likely in residents with lower initial depression scale scores, higher levels of activity, shorter stays in homes, and a history of previous psychiatric in-patient treatment. Of course, residents with lower depression scores needed to make a lesser improvement in order to achieve categorical recovery.

Higher activity levels might be causative of some improvement and the treatment plans implied a prediction that activity would benefit depressed residents. The fact that residents who recovered had been in homes a shorter time might imply some form of depression directly related to the experience of admission, which remits with time in at least a proportion of sufferers. It may also be taken to suggest that prolonged residence in a Part-III home eventually may sap the resilience of those with potentially recoverable depressive disorders. The significance of previous psychiatric in-patient treatment as a predictor of recovery is unclear, but it was not accounted for by a subgroup of residents with recurrent unipolar depressions, prone to remit and relapse over years.

The lack of an association between recovery and intervention status is unsurprising, given the low number of recommended interventions implemented, but there was no evidence that even the interventions which were delivered had any efficacy in relieving depression. In many cases the environment may have contributed to the maintenance of depressed mood, but the findings also were consistent with suggestions that pre-existing personality traits contribute to loneliness and depression in old age (Murphy, 1986).

The main factors predictive of death at one year were age, physical illness, and impairment of activities of daily living, but specific psychiatric diagnosis, degree of depression or dementia, and intervention status were not significant predictors of mortality. The causes of death in depressed residents were unremarkable for a population of this type. Death rates were notably higher than those recorded in earlier studies of somewhat younger elderly depressed in-patients and out-patients (Murphy, 1983; Baldwin & Jolley, 1986; Magni *et al*,1988), and also higher than those found in a large study of Part-III home residents in another part of Britain (Booth *et al*, 1983).

Conclusions

Depression is a common disabling phenomenon in residents of Part-III homes. It is likely to be associated with low levels of social intercourse, to be chronic, not to remit, and to be associated with high mortality. Attempts at therapeutic intervention with individual residents by one psychiatrist were not beneficial, but definitive conclusions about efficacy were not possible.

The depression syndromes encountered were not all severe, but they represented a large burden of suffering for a significant proportion of the population investigated, and their association with physical disorders and dependency implied a large demand on thinly stretched staff resources. There is a pressing need to determine what factors predict and precipitate these depressive conditions, and contribute to their maintenance among residents of Part-III homes. Meanwhile, it seems that usual environments in homes for the elderly are impoverished and lack stimulation (Godlove *et al*, 1982; Tarbox, 1983). Some are quite disgraceful (Southwark Council, 1987). Thus improvements in environments, with an increase in available activities, must be laudable aims. The assessment of the efficacy of these changes should be an aim of research.

A possible greater role for the psychogeriatric service in the management of Part-III homes is an important topic for further discussion. Perhaps Part-III homes would be better run on a collaborative model, with input from both local authorities and area health authorities. The Griffiths (1988) report with its recommendation that local authorities should be the sole care managers for the chronically mentally ill is not a promising start.

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Plasma N-POMC, ACTH and Cortisol Concentrations in a Psychogeriatric Population

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Elderly patients with depression and Alzheimer-type dementia (ATD) were compared with age-matched control subjects using a protocol which measured cortisol, adrenocorticotrophic hormone (ACTH) and N-terminal pro-opiomelanocortin (N-POMC) to determine diurnal variation and the effect of dexamethasone administration. Depressed patients had significantly elevated cortisol concentrations both before and after dexamethasone administration. Basal ACTH and N-POMC concentrations were normal in depressed patients but were both elevated, compared with controls, after dexamethasone. By contrast, in ATD patients, cortisol was elevated only after dexamethasone, as was ACTH, but not N-POMC. This may imply that the pattern of secretion of POMC-derived peptides underlying increased cortisol secretion is different in ATD from that in depression.

Pro-opiomelanocortin (POMC) is a glycoprotein precursor from which a family of peptides are derived, including adrenocorticotrophic hormone (ACTH), β-lipotrophin (β-LPH), β-endorphin (β-EP), and N-terminal pro-opiomelanocortin (N-POMC) (Lowry, 1984). N-POMC is a peptide of 76 amino acids, whose carboxy terminal sequence has been termed γ_3MSH (Benjannet *et al*, 1980). Plasma N-POMC shows a similar pattern of secretion to ACTH, with circadian variation, suppression by dexamethasone, and elevation in response to insulin hypoglycaemia and intravenous administration of corticotrophin-releasing hormone (CRH) (Chan *et al*, 1983; Hale *et al*, 1984; Motomatsu *et al*, 1984).

N-POMC may have a role in modulating adrenal growth and secretion. The γ_3MSH portion potentiates the steroidogenic effect of ACTH on adrenal cells *in vitro* (Pedersen *et al*, 1980). Animal studies have demonstrated that both the intact peptide and the γ_3MSH portion promote adrenal hypertrophy in synergy with ACTH. The amino terminal portion (1–48/49) promotes mitotic activity (Estivariz *et al*, 1982) and may have an important role in compensatory adrenal growth after unilateral adrenalectomy (Lowry *et al*, 1983). This proposed adrenal regulatory function has a possible bearing on the well established findings of elevated basal cortisol and cortisol resistance to dexamethasone suppression which occur in a substantial proportion of depressed patients (Sachar *et al*, 1973; Carroll *et al*, 1981).

We have previously reported that plasma levels of both ACTH and N-POMC are relatively resistant to dexamethasone suppression in depression (Charlton *et al*, 1987, 1988). This report extends these studies to elderly patients with depression and compares them with patients with Alzheimer-type dementia (ATD) and with age-matched controls. ATD patients are of interest because of reports of their high incidence of cortisol resistance to dexamethasone suppression (Raskind *et al*, 1982; McKeith, 1984).

Method

Ethical permission for this study was given by the Newcastle Health Authority Ethical Committee.

Depressed patients were selected from two psychogeriatric assessment units. DSM–III criteria for major depressive episode were employed (American Psychiatric Association, 1980). Subjects for this and other groups were excluded when physical illness or medication contraindicated a dexamethasone suppression test (Carroll *et al*, 1981). Severity of depression was measured using the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) and 'endogenicity' was assessed by the Newcastle Endogenous Depression Diagnostic Inventory (NEDDI; Carney *et al*, 1965).

Subjects with ATD were in-patients from two psychogeriatric assessment units. They had been screened by psychogeriatricians and patients with a clinical profile suggestive of arteriosclerotic dementia were excluded. DSM–III criteria for primary degenerative dementia were employed. Patients were excluded if depressive symptoms were prominent.

Control subjects consisted of three groups: orthopaedic in-patients awaiting minor elective surgery for osteoarthritis, healthy volunteers, and convalescent in-patients from medical wards. Subjects had no current psychiatric symptoms and no history of psychiatric illness.

Blood samples were collected in lithium-heparin-coated tubes which were maintained at 4°C during collection and centrifugation. Enzyme inhibitors were added to prevent peptide degradation (Charlton *et al*, 1987).

N-POMC (1–76) was measured using an immunoradiometric assay ('IRMA'; Charlton *et al*, 1988). Minimum detectable concentration was 20 pg/ml and the interassay variation was 14%. ACTH was also measured using an IRMA (Hodgkinson *et al*, 1984). Minimum detectable concentration was 10 pg/ml and the interassay variation was 13%. Cortisol was measured using a modification of the competitive protein-binding assay (Murphy, 1967). Minimum detectable concentration was 40 nmol/l and the interassay variation was 9%.

Samples were taken at 09.00 h and 23.00 h on day 1, after which 1 mg of dexamethasone was administered orally at 23.00 h and a further sample was collected at 16.00 h on day 2 from all subjects. Since the main findings of the serial sampling study concerned the post-dexamethasone results, samples were taken at 16.00 h on day 2 from a further six controls, four depressives, and four ATD patients. These additional patients were chosen to make three groups that were closely matched in terms of age and sex.

Statistics

The distribution of the data was skewed (Filliben's test), but Grubbs' test revealed no significant outliers. Plots of means versus standard deviations showed strong linear trends for each hormone and, as recommended by Zivin & Bartko (1976), log transformation was therefore carried out. Filliben's testing of the transformed data revealed normal distributions. Transformed data were analysed by one-way analysis of variance (ANOVA). Where the *F* ratio showed a significant group difference, Neuman-Keul's test was applied.

Results

There were 11 patients with major depression (3 men, 8 women, mean age 75 ± 1.9 years, range 66–84), 11 ATD patients (2 men, 9 women, mean age 77 ± 1.9 years, range 69–87) and 9 controls (3 men, 6 women, mean age 73 ± 1.6 years, range 67–82). The age and sex ratios of these groups do not differ significantly. This was also true when the groups were expanded, for further post-dexamethasone results, to 15 depressed patients (5 men, 10 women, mean age 76 ± 1.7 years, range 66–86), 15 ATD patients (5 men, 10 women, mean age 76 ± 1.6 years, range 69–87), and 15 controls (5 men, 10 women, mean age 74 ± 1.5 years, range 67–86).

Seventy-three per cent of the depressed patients were classified as having major depression with melancholia and 91% had 'endogenous' depression (score of >6 on NEDDI). The HRSD scores were in the range 17–44. Four patients were drug free but six were taking antidepressants, three major tranquillisers, and two were taking both. Two patients were also taking tricyclic medication. In the ATD group four patients were drug free and the remainder were taking medication for coincidental physical illness. The controls were all drug free. In the expanded study of hormone levels after dexamethasone, the four additional depressed patients had HRSD scores in the range 20–40, and all were classified as major depressives with melancholia and 'endogenous-type' depression.

Cortisol, ACTH and N-POMC results from the first groups are displayed in Table I. One-way ANOVA revealed significant group differences in cortisol concentrations ($F(2,31) = 14.29$, $P = 0.00012$). Cortisol concentrations in depression patients were significantly ($P < 0.05$) increased when compared with controls at 23.00 h on day 1 and 16.00 h on day 2. Cortisol was significantly ($P < 0.05$) elevated in the ATD patients compared with controls at 16.00 h on day 2. There were no significant differences between depressed and ATD patients.

ANOVA revealed significant group differences in ACTH and N-POMC concentrations ($F(2,31) = 6.47$, $P = 0.0048$; $F(2,31) = 3.39$, $P = 0.048$), respectively. Levels of ACTH and N-POMC were not significantly altered by dexamethasone in either the depressed or ATD patients. Both groups of patients had significantly ($P < 0.05$) elevated ACTH when compared with the control group at 16.00 h on day 2. N-POMC was significantly ($P < 0.05$) elevated at this time in depressed, but not ATD, patients compared with controls.

For the expanded groups (Table II), ANOVA revealed significant group differences in cortisol, ACTH, and

TABLE I

Mean (±s.e.m.) concentrations of plasma hormones

	Time sample taken	*Controls (n = 9)*	*Patients with major depression (n = 11)*	*Patients with Alzheimer -type dementia (n = 11)*
Cortisol:	09.00	430 ± 28	534 ± 43	616 ± 92
nmol/l	23.00	127 ± 24	329 ± 29*	235 ± 43
	16.00^1	72 ± 15	273 ± 42*	241 ± 61*
ACTH:	09.00	38 ± 11	44 ± 2.7	49 ± 6.4
pg/ml	23.00	26 ± 6.4	40 ± 3.6	35 ± 3.2
	16.00^1	19 ± 4.2	38 ± 4.4*	36 ± 5.1*
N-POMC:	09.00	182 ± 26	146 ± 27	152 ± 28
pg/ml	23.00	104 ± 22	114 ± 23	85 ± 15
	16.00^1	73 ± 16	120 ± 19*	96 ± 22

*$P < 0.05$ compared with controls.
1. On day 2, after administration of dexamethasone.

TABLE II

Mean (±s.e.m.) concentrations of plasma hormones after administration of dexamethasone

	Controls (n = 15)	*Patients with major depression (n = 15)*	*Patients with Alzheimer-type dementia (n = 15)*
Cortisol: nmol/l	59 ± 10	234 ± 38**	231 ± 47**
ACTH: pg/ml	17 ± 5.3	38 ± 4*	36 ± 5.4*
N-POMC: pg/ml	60 ± 11	108 ± 15*	100 ± 20

* $P < 0.05$ compared with controls.
**$P < 0.01$ compared with controls

N-POMC concentrations at 16.00 h on day 2 ($F(2,42) = 21.38$, $P = 0.00001$; $F(2,42) = 19.19$, $P = 0.00002$; $F(2,42) = 3.72$, $P = 0.032$, respectively). Cortisol was significantly elevated in depressed and ATD patients compared with controls ($P < 0.01$ in both cases). ACTH was significantly elevated in depressed and ATD patients compared with controls ($P < 0.05$ in both cases). N-POMC was elevated, compared with controls ($P < 0.05$), in the depressed patients. A comparable but non-significant trend was seen in ATD patients compared with controls. No significant differences between depressed and ATD patients were detected for any of the hormones. No effect of tricyclic medication was seen.

Discussion

Several abnormalities of hormone concentrations are described but in all cases there is an overlap between control subjects and the psychiatric diagnostic groups.

Cortisol was substantially elevated in the depressed patients compared with controls at 23.00 h, and at 16.00 h, after dexamethasone administration. Basal concentrations of ACTH and N-POMC were normal. However, both plasma ACTH and N-POMC are elevated compared with controls after dexamethasone, which constitutes good evidence of relative resistance of POMC-derived peptides to steroid feedback in depression. A similar degree of relative resistance to dexamethasone has been reported for other POMC family members (e.g. β-endorphin, β-lipotrophin) (Brambilla *et al*, 1981; Matthews *et al*, 1982) although some studies have produced contrary results (Cohen *et al*, 1984; Ball *et al*, 1987). The dissociation of basal cortisol and ACTH concentrations has previously been reported (Kalin *et al*, 1982; Reus *et al*, 1983; Charlton *et al*, 1987).

One reason for this lack of correlation may be the pulsatile nature of hormone secretion, as single samples may give widely varying values according to whether sampling comes at, or between, peak levels. Further reasons could include the effect of differences in plasma half-life and the combined error due to the coefficients of variation in the assays. Clarification of this question would require a detailed study with frequent blood sampling and simultaneous measurement of all three hormones.

Nevertheless, these results suggest that in depression there are elevated levels of cortisol but normal levels of both ACTH and N-POMC. This implies that adrenal responsiveness to ACTH is increased in depression and this phenomenon has been demonstrated in studies where synthetic ACTH (1–24) has been administered to depressed subjects (Amsterdam *et al*, 1983; Jaeckle *et al*, 1987). It must be emphasised that the evience for an adrenal growth-promoting role of N-POMC is derived from *in vitro* and animal studies, and there is no direct evidence from humans. The present results show that basal N-POMC is not elevated in depression but that it shows relative insensitivity to suppression by both endogenous and exogenous glucocorticoids. Whether this is a factor in the maintenance of adrenal growth and ACTH sensitivity in depression remains speculative. It is noteworthy that while the differences in cortisol between the depressed patients and the controls were large and statistically robust, the changes in pituitary hormones after dexamethasone were less so. The changes in ACTH levels occupy an intermediate position but the changes in N-POMC are just below the limit of conventional significance ($P < 0.05$) and this indicates the need for a larger replication study.

The ATD patients had elevated cortisol concentrations after but not before dexamethasone. This pattern is similar to that seen in previous studies (Raskind *et al*, 1982; Davis *et al*, 1986). There was no significant elevation of ACTH in basal samples, which confirms our previous report using a separate group of patients (Ferrier *et al*, 1988). N-POMC concentrations were normal at all sampling times. This finding could imply that the mechanism of secretion of excess cortisol is different in ATD from that in depression. Little research has been published on the detailed examination of the hypothalamo–pituitary–adrenal axis in ATD: further studies would be worthwhile.

The effects of psychotropic drugs are a confounding factor. While tricyclic medication by itself does not seem to account for the cortisol abnormalities, the variable effects of these drugs may be important in studies aimed at determining pituitary and adrenal sensitivity. It is clear that the relationship between cortisol, ACTH, and N-POMC is complex, and requires elucidation both in terms of normal physiology and within a variety of psychiatric illnesses.

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Responses of Growth Hormone to Desipramine in Endogenous and Non-endogenous Depression

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Desipramine, a monoamine reuptake inhibitor, was used to stimulate release of growth hormone (GH) in 29 DSM–III major depressives and in 10 healthy controls. Eighteen of the depressives showed a blunted response. The GH-stimulation test was unable to distinguish endogenous from non-endogenous patients. The 13 dexamethasone non-suppressors were more likely to have a blunted GH response than the 14 suppressors. The results indicate that at least a subset of non-endogenous depressives have significant neuroendocrine abnormality.

The most widely studied test of central noradrenergic alpha-2 receptor functioning in man is the clonidine/growth-hormone (GH) challenge test. These alpha-2 adrenoceptors are located both pre- and post-synaptically and research in animals indicates the GH response is mediated by post-synaptic forebrain receptors. Lovinger *et al* (1976) found that the response was blocked in dogs when the alpha-2 antagonist phenoxybenzamine is injected into the third but not the fourth ventricle. The work of Katakami *et al* (1984) indicates that the bulk of forebrain alpha-2 adrenoceptors involved in the control of GH release are located in the arcuate nucleus, where GH-releasing factor is produced.

Several groups have found a strong relationship between GH blunting and endogenous features of depression (Matussek *et al*, 1980; Checkley *et al*, 1981, 1984; Charney *et al*, 1982; Siever *et al*, 1982). Paykel's group failed to replicate this, finding equal levels of blunting in endogenous and non-endogenous depression (Katona *et al*, 1986). They however found a link between blunting and dexamethasone non-suppression.

Clonidine is an alpha-2 agonist which, in stimulating receptors, brings about the release of GH. As a test the clonidine/GH response is relatively easy to conduct but it can be unpleasant for the patient. It involves the intravenous administration of clonidine in doses of 0.5–5.0 μg/kg body weight.

Monoamine reuptake inhibitors also increase GH levels, by blocking the reuptake of noradrenaline and thus indirectly stimulating the alpha-2 adrenoceptor. Desipramine and maprotiline, which act predominantly on the noradrenergic system, produce the greatest increase in GH levels, while clomipramine, which acts mainly on the serotonin system, has little effect (Sawa *et al*, 1982). The effect of desipramine is blocked by the alpha-1 and alpha-2 antagonist phentolamine but not by the alpha-1 antagonist prazosin or the 5-HT antagonist methysergide (Laakmann *et al*, 1986).

When taken orally in healthy subjects 100 mg of desipramine causes a significant increase in GH levels after a delay of around 150 minutes (Laakman *et al*, 1977). On seven healthy volunteers we carried out both clonidine/GH and desipramine/GH tests and all reported greater subjective distress from the former test (unpublished). Thus, on the basis of patient acceptability, the desipramine test may provide a suitable alternative to the clonidine test.

It has been reported that neurotically (non-endogenously) depressed patients stimulate GH secretion more strongly than psychotically (endogenously) depressed patients (Sawa *et al*, 1981) and blunted responses in endogenous depression have been reported by Glass *et al* (1982), Calil *et al* (1984) and Meesters *et al* (1985). We decided to examine in greater detail the characteristics of this test in depressed patients, since it is relatively easy to conduct and free from side-effects (in relatively low doses), apart from very mild sedation. In particular we examined its capacity to distinguish between endogenous and non-endogenous forms of depression.

Method

Twenty-nine physically healthy patients (mean age 41.8 ± 14.9 years) with a diagnosis of DSM–III major depression (American Psychiatric Association, 1980) were included in the study. Patient characteristics are described in detail in Table I. Nineteen were completely drug free on entering the study and the remainder had medication discontinued for at least two weeks (except for temazepam, which was allowed as a hypnotic). Twenty were endogenously depressed and nine were non-endogenously depressed as judged by the Newcastle scale for endogenicity (Carney *et al*, 1965). All had scores of 18 and over on the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967).

TABLE I
Characteristics of the 29 major depressives

Patient	*Age: years*	*Sex*	*Diagnosis*	*Score on HRSD*	*Score on Newcastle scale*	*GH response: mU/l*	*DST suppression*
1[1]	58	F	P,BP	43	4	0	+
2	28	F	M,UP	25	5	5.2	+
3	41	M	M,BP	22	10	0	−
4[1]	20	F	M,UP	33	7	15.5	−
5	59	M	M,UP	30	8	0	+
6	65	F	P,UP	31	5	0	−
7	44	M	M,UP	19	6	12.7	+
8	60	F	M,UP	23	7	0	+
9[1]	41	M	UP	19	4	10.9	+
10[1]	33	F	M,UP	24	8	13.4	+
11	36	M	M,UP	30	7	46.6	+
12[1]	38	F	M,UP	29	9	0	−
13	29	F	P,BP	38	10	3.2	Not done
14	55	F	M,UP	32	8	9.7	−
15[1]	30	F	M,UP	18	4	0	+
16	30	F	P,BP	38	4	10.9	+
17	26	F	M,UP	31	4	6.5	−
18	30	M	P,UP	36	9	21.5	−
19[1]	70	F	M,UP	29	4	0	+
20	52	F	M,UP	30	5	33	+
21[1]	29	M	M,UP	25	10	16.9	+
22	30	M	M,UP	27	10	4.8	−
23[1]	25	M	M,UP	31	7	3.2	−
24[1]	73	F	M,UP	34	8	0	−
25	36	F	P,BP	28	10	0	−
26	43	F	M,UP	30	7	0	Not done
27	43	F	M,UP	35	12	3.5	−
28	29	F	UP	36	6	3.3	+
29	69	M	M,UP	34	9	2.2	−

1. Patient drug free for at least two weeks.
P = psychotic; M = melancholic; UP = unipolar; BP = bipolar.

Ten healthy subjects acted as controls. None of these had a history of psychiatric illness. The mean (± s.d.) age of this group was 34 ± 10.1 years (range 26–54 years).

Patients and controls presented at 8.30 a.m. having fasted from midnight. An 18G cannula was inserted into a forearm vein. The patient was allowed to relax for 20 minutes before blood was taken for estimation of GH. Two baseline 8 ml samples were collected 15 minutes apart in lithium-heparin tubes. Subjects rested during the test but were prevented from sleeping. An oral dose of desipramine (1 mg/kg body weight) was given (this was rounded to the nearest 25 mg so that, for example, a 90 kg man received 100 mg). Blood for further GH estimation was collected 90, 120 and 180 minutes after the oral desipramine. The GH was assayed by a double antibody radioimmunoassay (Diagnostic Products Corporation method; Salvatore Raite, 1983). Subjects with baseline GH levels greater than 3 mU/l were not included in the analysis. Responses were regarded as blunted if GH levels failed to rise at least 8 mU/l above baseline values.

Where possible patients also had a dexamethasone suppression test (DST), two to three days before the GH stimulation test, using the method described by Carroll (1982). They were given 1 mg dexamethasone at 10 p.m. and blood was taken for cortisol estimation at 4 p.m. the next day. A failure of dexamethasone to lower cortisol to below 137 nmol/l was used as the definition of non-suppression.

Results

The GH responses to the desipramine challenge were considerably blunted in the depressives. For 18 of the depressives GH levels rose less than 8 mU/l above baseline (Fig. 1). The change in GH level was taken as the difference between the mean baseline value and the maximum value after desipramine.

Changes in GH levels differed significantly between patients and controls (unpaired t-test: $t=2.17$, d.f. $=37$, $0.05>P>0.02$). When patients with an endogenous depression are compared with those non-endogenously depressed no significant difference in the mean GH response is seen (unpaired t-test: $t=0.10$, d.f. $=27$). Twelve of the endogenously depressed patients and six of the nine non-endogenously depressed patients had blunted responses (change in GH level <8 mU/l). The test does not therefore

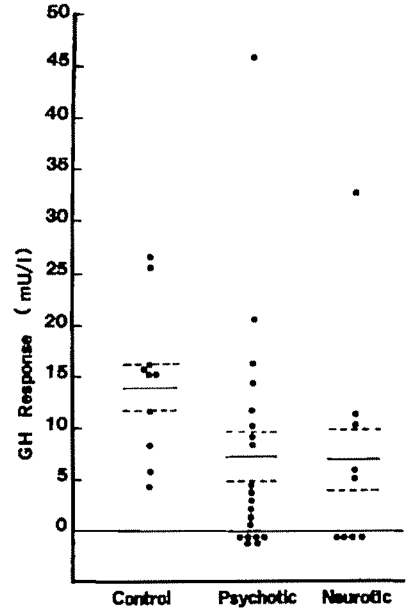

FIG 1. Growth-hormone responses to desipramine in control subjects and in psychotic (=endogenous) and neurotic (=non-endogenous) depressives. Responses refer to the maximum GH level after desipramine challenge relative to the baseline estimate. The mean (solid line) ± s.e.m. values (dashed lines) for each group are shown. Values under the line indicate subjects in whom there was no detectable response to desipramine.

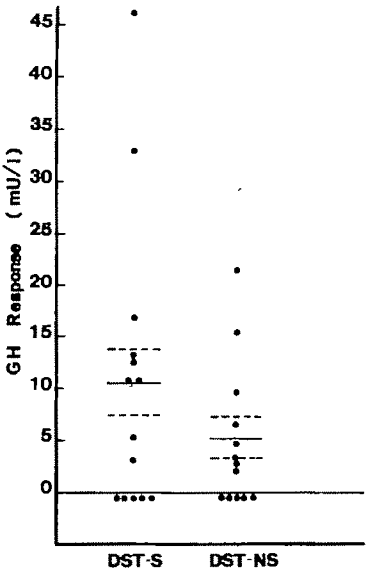

FIG. 2 Growth-hormone responses to desipramine in dexamethasone suppressors (DST-S) and non-suppressors (DST-NS). Responses are calculated as the maximum GH level after desipramine challenge relative to the baseline estimate. The mean (solid line) ± s.e.m. values (dashed lines) for both groups are shown. Values under the line indicate subjects in whom there was no detectable response to desipramine.

distinguish between these groups. When severity of depression (HRSD score) is correlated with the GH response again no relationship emerges ($r = -0.01$). Neither is there a significant relationship between the response and the endogenicity score ($r = -0.06$) or the patients' age ($r = -0.25$).

The DST non-suppressors had a lower mean GH response than the DST suppressors but this did not reach statistical significance (unpaired t-test: $t = 1.39$, d.f. = 25). Overall 10 of 13 non-suppressors had blunted responses, as did 7 of 14 suppressors (Fig. 2). This difference in distribution is significant ($\chi^2 = 4.23$, d.f. = 1, $0.05 > P > 0.025$).

Discussion

The present results are in keeping with previous reports of blunted GH responses in depressed patients (Matussek *et al*, 1980; Checkley *et al*, 1981; Charney *et al*, 1982; Siever *et al*, 1982; Sawa *et al*, 1982). Checkley *et al* (1984), using the Newcastle scale to distinguish endogenous from non-endogenous depression, found significant differences in GH response. Using the same instrument we were unable to demonstrate such a difference. The only other negative reports are those of Dolan & Calloway (1986) and Katona *et al* (1986). In keeping with previous work we were unable to find any relationship between the severity of depression as measured by the HRSD and the GH response. When patients with psychotic symptoms were compared with those without, no differences were detected. Furthermore when patients on the benzodiazepine temazepam were compared with entirely drug-free patients no differences were detected. Neither could blunted responses be explained in terms of illness duration, as patients with relatively short episodes of depression were no less likely to show blunting.

Methodologically the essential difference between our study and that of Checkley *et al* (1984) is the fact that Checkley's group used the alpha-2 agonist clonidine intravenously to stimulate GH release directly, whereas we used the monoamine reuptake inhibitor desipramine, which, as Laakmann *et al* (1986) have shown, indirectly stimulates the alpha-2 adrenoceptor, bringing about GH release. It is unlikely that this methodological difference would account for the difference in results, although the intravenous route does overcome potential complicating pharmacokinetic variables. Laakmann (1980) and Sawa *et al* (1981), also using desipramine as the challenge drug, found significant differences between endogenous and non-endogenous depressives. A second difference between our study and that of

Checkley *et al* (1984) is the method of assessing GH response: we measured this as the peak GH level above baseline, whereas Checkley's group used the area under the curve. Again this is unlikely to account for the present differences. Ansseau *et al* (1988), calculating the GH response by both the peak method and the area under the curve, found that both methods gave similar results.

The DST is undoubtedly the most widely studied neuroendocrine tool in psychiatry (for review see Carroll, 1982). The frequency of abnormal DST results in depression varies widely from one study to another (Holden, 1983). Of 27 patients tested in our study 13 (48%) were DST non-suppressors. DST non-suppressors were more likely to have blunted GH responses than suppressors, although differences in the means of both groups did not reach statistical significance. Our results are in keeping with those of Katona *et al* (1986), who also found that non-suppressors were more likely to show blunting of GH response. Five of our sample classified as non-endogenous on the Newcastle scale had blunted GH responses and two of these were also DST non-suppressors. This indicates that at least a subset of patients with non-endogenous depression have significant biological changes. Our results also indicate that the desipramine/GH stimulation test, conducted using the oral route of desipramine administration, may be more sensitive for detecting cases of major depression than the DST.

Nineteen of the patients entering this study were entirely drug free; the other ten had washout periods of at least two weeks. The duration of the washout was dictated by clinical circumstances and ethical considerations but, as Corn *et al* (1984) and Checkley *et al* (1986) have pointed out, such a washout may be inadequate. Nonetheless, 'washout' patients were evenly distributed between the endogenous and non-endogenous groups and overall the responses of these ten patients did not differ from those of the remainder of the sample.

Laakmann *et al* (1977) report convincing evidence that the action of desipramine in elevating GH is mediated via the noradrenergic system. If such is the case the current results provide further support for the theory of noradrenergic dysfunction in at least a subgroup of depressed patients (Maas, 1975). The results are consistent with a functional down-regulation of post-synaptic noradrenergic receptors perhaps secondary to a hyperadrenergic state in depression. Alternatively, reduced pre-synaptic availability of noradrenaline would decrease the effect of desipramine. The first of these hypotheses is more consistent with the blunted GH responses seen following clonidine administration.

In summary we can find little relationship between blunted GH responses to desipramine and the traditional classification of depression into endogenous and non-endogenous subtypes. A relationship does exist between GH responses and DST status.

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The Stress Profile*

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The Stress Profile consists of a number of questionnaires which have been developed to assess the severity of stress in nine areas of life: social habits, social relationships, life events, sexual problems, sleep, psychiatric symptoms, old age, menstrual stresses, and 'stress and the heart'. The questionnaires have been validated for inter-rater reliability and against the Hamilton anxiety and depression rating scales Using the Profile, the inter-relationships between different stress areas can be assessed, appropriate treatments advised, and the response to treatment in relation to different components evaluated.

The Maudsley Stress Clinic was established in order to diagnose and quantify the effects of stress on the health and well-being of those referred to it (Wheatley, 1984). The Stress Profile was developed for the same purpose. It consists of questionnaires in nine different stress areas, the answers being quantified and subsequently represented diagrammatically (Wheatley, 1987*a*). The Profile can be used also for recording subsequent response to therapy (Wheatley, 1987*b*). Although references to its use have been made in various reports, it has not been described in detail. It consists of the following sections: social habits, social relationships, life events, sexual problems, sleep, psychiatric symptoms, old age, menstrual stresses, and 'stress and the heart'.

Each section is divided into a number of subitems, which are scored individually for severity. The total score in each area is not necessarily the same, so that the resultant Stress Profile is constructed in relation to the proportional scores of the constituent sections. An example Stress Profile of a patient with major depression is shown in Fig. 1.

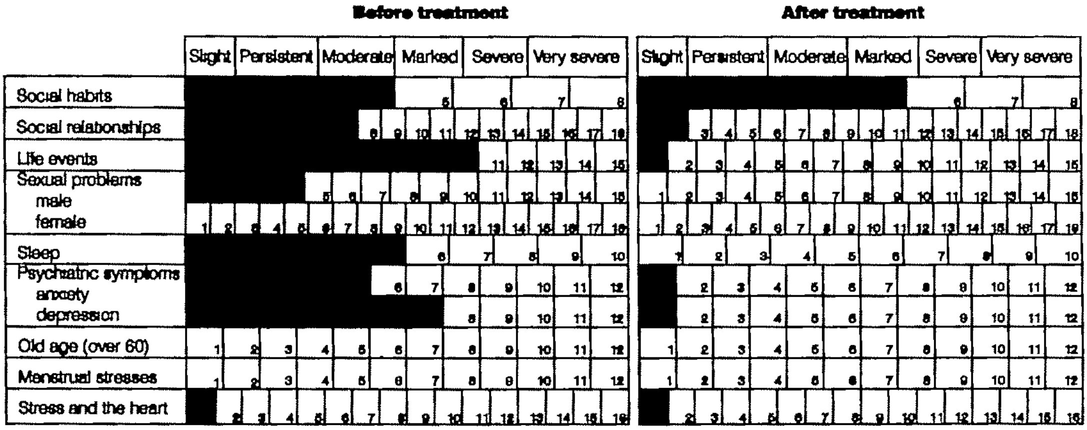

FIG. 1 Stress Profiles of a patient (under 60 years) with major depression (DSM-III-R) before and after 12 weeks' treatment with antidepressant drug therapy, showing reductions in severity of many stress areas, with the exception of social habits and 'stress and heart' (dark areas indicate patient's scores on each item).

Stress areas

Social habits

This records the patient's use of alcohol, tobacco, caffeine-containing drinks, and addictive drugs. With the exception of alcohol, each of these sections is scored as none (0), light (1), or heavy (2), giving a maximum score of eight for this section. For alcohol the scores are: none (1), light (0) and heavy (2), since there is evidence that light alcohol consumption is beneficial, particularly in relation to the cardiovascular system (Gill *et al*, 1986; Shaper *et al*, 1987). The divisions between 'light' and 'heavy' relate to daily consumption: three drinks (Anderson, 1976), 15 cigarettes and five cups respectively. For addictive drugs, 'light' corresponds to irregular use (parties, weekends, etc.) and 'heavy' to regular use.

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*Copies of this instrument are available from DW for a sum of £1 (UK) or £3 (overseas) (US$5) to cover production and postal costs.

Social relationships

This consists of six items, each of which is scored on a four-point scale (none (0), light (1), moderate (2), considerable (3)), giving a maximum score of 18 for the whole section. The individual items are concerned with: discontent at work or due to unemployment; mental strain in recreations; marital problems; lack of a close confidante; relationships with other people and opportunities for social contacts; and problems concerning physical appearance.

Life events

The questionnaire is divided into five items concerned with different types of events that have been found to influence the response to psychotropic drugs (Wheatley, 1982): emotional, living (earning), living (enjoyment), general health, and serious illness or death. Each item is scored on a four-point scale relating to the degree of stress still experienced by the patient by the event: none (0), slight (1), moderate (2), very much (3). Stress may be caused by both beneficial and adverse events, although more frequently the latter. The maximum score is 15 for this section.

Examples of beneficial emotional events are: love relationships, engagements, marriage, birth of babies; and adverse ones are: broken romances, separation, unwanted pregnancies, abortion, etc. Living (earning) events include: promotion at work, passing examinations, financial gains; dismissal, failing examinations, financial losses, demotion, etc. Living (enjoying) events include: acquisition of prized objects, parties, outings, happy world events; damage in the home, car accidents, losing at sport, etc. General health events may involve the patient or others: recovery from illness, successful operations; recurrent coryza, bronchitis, arthritis. Serious illness or death refers to a close friend or relative or the patient for the former.

It is important to highlight for the patient the difference between social relationships, which refer to the present time, and events, which refer to the past, although they may be of a similar nature.

Sexual problems

There are five main items and one extra for women, each item being scored on a four-point scale (none (0), slight (1), moderate (2), very much (3)), giving a possible total of 15 for men and 18 for women. The items are: dissatisfaction with degree of libido; stress in sexual relationships or lack of them; physical problems such as poor erection, premature ejaculation, anorgasmia, etc.; guilt feelings; and stress in relation to masturbation. The additional item for women is concerned with dysparunia and fear of pregnancy.

Sleep

The experience of sleep is assessed on five characteristics, each one being rated 0–2, giving a maximum total of 10. These items are: delayed onset of sleep (< ½ hour (0), ½–1 hour (1), > 1 hour (2)); nocturnal awakenings (0–1 (0), 2–3 (1), ⩾4 (2)); early morning waking (< ½ hour (0), ½–1 hour (1), > 1 hour (2)); average hours of sleep (7–9 (0), 5–6 (1), < 5/9 > (2)); and feeling refreshed on waking (completely (0), moderately (1), not much (2)).

Psychiatric symptoms

The Raskin depression (1970) and Covi anxiety (1974) scales are completed to indicate overall psychiatric morbidity. Each scale consists of three parts scored on a five-point scale (none (0), mild (1), moderate (2), severe (3), and very severe (4)) giving a maximum score of 12. The Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967) and for Anxiety (HRSA; Hamilton, 1959) are also completed, as indicated later.

Old age

There are four items: degree of unhappiness with the way of life; problems with deafness or defective vision; impact of other physical defects (arthritis, etc.); and dissatisfaction at not being able to do things that were previously possible. Each item is scored as none (0), slight (1), moderate (2) and very much (3), giving a maximum of 12.

Menstrual stresses

The items here include menstrual abnormalities, and either pre-menstrual symptoms or menopausal effects, the last two being themselves subdivided into three sections. Pre-menstrual syndrome (PMS), defined as symptoms occurring or accentuated any time during 14 days before period and/or first 1–2 days of period, includes: anxiety and depression; irritability, loss of energy and difficulty in thinking; and physical symptoms such as weight gain, swollen breasts, etc. Menopausal symptoms, defined as occurring from age 35 onwards, with or without menstrual changes (heavy, missed, irregular periods) or following hysterectomy include: physical (hot flushes, sweats, headaches, etc.), sexual (reduced libido, dry vagina, etc.) and affective (anxiety and depression). Each item (or section) is scored on a four-point scale (none (0), slight (1), moderate (2), severe (3)), giving a maximum score of 12.

Stress and the heart

This section is scored on four items using five-point scales (0–4), giving a possible total of 16. Family history (heart trouble, hypertension, diabetes etc.) is scored according to the number of relatives having one or more of these disorders. (no relatives (0)–four or more relatives (4)). The diastolic level, assessed (sitting) at interview (95–115 mmHg (1), 116+ mmHg (2)) is combined with pulse rate (100–120 (1) and 120+ or irregular (2)), to give the second item, which has the same possible maximum score (4) as the others. Coronary heart disease is scored progressively as: suspicious pain on exertion (1), undue shortness of breath on exertion (2), ECG proven (3) and previous myocardial infarction (4). Finally, cardiophobia is assessed according to the worry caused: slight (occasional) (1), moderate (when not otherwise mentally occupied) (2), marked (most of the time) (3) or extreme (dominating patient's life) (4).

Validation

Inter-rater reliability

Ratings were undertaken consecutively by two different observers at the patient's first attendance at the clinic in a sample of 24 cases, and correlation coefficients were calculated for each stress area and the total profile score. The only exceptions were 'stress and the heart' and 'old age', which were not analysed. In the case of the former, one observer was not skilled in taking cardiovascular measurements, and in the case of the latter, there were too few patients aged over 60 years for analysis.

As shown in Table I, agreement was good for the individual sections (0.69 (depression)–1.0 (social habits)). The coefficient for the total profile scores was 0.91. All these coefficients were highly statistically significant ($P<0.001$).

Hamilton ratings

There is no independent measure of stress with which the Stress Profile can be compared and indeed stress-induced illness is such a relatively new concept that comprehensive stress scales have not yet been developed. However, there are precedents for using the HRSA and HRSD for comparative purposes (Carroll *et al*, 1981). While these scales are not measures of stress *per se*, anxiety and depression have been the most important manifestations of the response to stress seen in the clinic (Wheatley & Lader, 1987). Therefore the total profile scores were correlated with the HRSA and HRSD totals in a sample of 95 patients, irrespective of diagnosis. Furthermore, correlations were calculated in 28 of these cases at the end of treatment.

As shown in Table II, correlations were not as close as in the case of inter-rater reliability, but were still highly significant in respect of both comparisons ($P<0.001$).

TABLE I
Inter-rater reliability in relation to Stress Profile sections and total severity scores

Stress item	*Mean score (n = 24)* observer A	observer B	*Correlation coefficient (r)*
Social habits	2.8	2.8	1.0
Social relationships	4.7	5.0	0.84
Life events	5.5	5.6	0.78
Sexual problems	1.3	1.5	0.95
Sleep	2.4	2.3	0.88
Psychiatric symptoms			
anxiety	5.1	5.2	0.87
depression	4.2	4.6	0.69
Old age	—	—	—
Menstrual stresses	1.4	1.6	0.88
Stress and the heart	—	—	—
Total	26.9	28.1	0.91

TABLE II
Correlations between total scores on the Stress Profile and those on the Hamilton scales

	Before treatment (total scores) (n = 95)			*After treatment (total scores) (n = 28)*		
	Stress Profile	*HRSA*	*HRSD*	*Stress Profile*	*HRSA*	*HRSD*
Mean	34.3	22.7	20.7	22.8	14.2	12.5
s.e.m.	1.5	0.7	0.7	2.2	1.4	1.5
r	—	0.48	0.61	—	0.63	0.62
$P<$	—	0.001	0.001	—	0.001	0.001

Indeed, had the correlations been extremely high it might have suggested that the Stress Profile is redundant since anxiety and depression might be rated instead.

Further correlations were made between the two main diagnostic categories (anxiety/panic/phobias and depression) and the total Hamilton scores. Although there were similar significant correlations of the diagnostic categories to both Hamilton scales, there was no higher correlation between specific diagnosis and specific scale. This probably underlines the fact that there is considerable symptom overlap between the two Hamilton scales and that the patients attending the clinic suffered from multiple stress problems with an overlay of anxiety and somatic symptoms.

Cardiovascular measures

As yet, only 15 patients have had cardiovascular measurements taken before and after treatment, irrespective of diagnosis. There were reductions in mean pulse rate, and in systolic and diastolic blood pressures, these changes reaching significance in the case of the blood-pressure measurements ($P<0.01$ and <0.02 respectively). No correlations were found between these reductions in haemodynamic measures and relief of total stress in these patients.

Discussion

The Stress Profile can be administered by a social assistant at interview or even self-recorded by the patient. The psychiatrist can then amplify the data either by further elucidation of the salient points or by using more comprehensive scales, which may be standard instruments or others that have been further devised for use in the clinic (Wheatley, 1984). Significant improvement in many items of the profile has been demonstrated with antidepressant drugs when depression was a major response to stress (Wheatley, 1990); and with beta-adrenergic blocking drugs in the case of anxiety and panic attacks (Wheatley, 1988). The Stress Profile provides a convenient means of assessing the effects of stress and diagnosing the main aetiological factors involved.

Acknowledgement

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The Friendships and Recent Life Events of Anxious and Depressed School-Age Children

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The number of moderate to severe undesirable life events and the quality of friendships were compared between a consecutive series of 100 childen aged 7–16 years with emotional disorders and a series of community controls matched for age, sex, and social class. Undesirable events and friendship difficulties exerted independent direct effects of similar magnitude on the probability of developing emotional disorders; 33% of disturbed children experienced both adversities compared with 6% of controls. An additive interaction was found for the two factors. The results were uninfluenced by the sex or pubertal status of the subjects. There appears to be no greater probability of being anxious rather than depressed in the presence of life events, alone or in combinations with friendship difficulties. Recent undesirable life events and friendship difficulties arise through independent social pathways and may exert their effects through different psychological mechanisms.

In recent years a number of studies have investigated the pathological effects of relatively long-standing social adversities in the lives of children and adolescents (Rutter, 1985). The risk of psychiatric disorder following more acute adversities has received less attention (Rutter, 1981). A recent investigation has confirmed that moderately or highly undesirable recent life events exert potentially causal effects on the onset of emotional and behavioural symptoms in school-age children and adolescents (Goodyer *et al*, 1985, 1987). The study also demonstrated that recent major events are neither necessary nor sufficient to provoke symptoms: 30% of the children did not experience a life event in the 12 months before onset of disorder; 20% of controls reported one or more major life events in the 12 months before interview.

It is apparent that a range of life experiences may potentially cause psychiatric disorders. Examples include long-term continuous difficulties such as poor relations with parents or parental psychiatric disorder (Rutter, 1990), life events such as bereavement or divorce (Van Eerdewegh *et al*, 1982; Hetherington *et al*, 1985), and recent adversities such as peer-group difficulties (Goodyer *et al*, 1989). The present evidence suggests that children with psychiatric disorder, particularly conduct symptoms, have experienced chronic social adversities. It may be the patterning and multiplicity of adversities that determines the risk of psychiatric disorder rather than the effects of any one type of event or difficulty (Rutter, 1987).

We have therefore considered in more detail other social factors that may be causally involved in the onset of anxious and depressive disorders of school-age children. We decided to continue our search for factors in the children's recent lives before examining the contributions of past experiences. One factor was the quality of childrens' friendships. The importance of friendships for well-being in childhood has been recognised by clinicians and researchers alike (Hartup, 1983; Rutter, 1985). Two broad themes are apparent from the literature. First, good peer relationships are probably necessary for healthy mental development. Thus they may operate by promoting a child's self-esteem and the development instrumental skills and emotional ties with other children (Hartup, 1983; Harter, 1983; Field *et al*, 1984). Second, the absence of close relationships with others may increase the risk of psychiatric disorder (Rutter, 1987).

Our previous research had shown that reports of peer-group difficulties as life events were uncommon (Goodyer *et al*, 1985). The impression was however that a sizeable proportion of children experienced peer-group problems as protracted difficulties over time rather than as discrete life events. Such difficulties were excluded from analysis because they failed to meet criteria for a circumscribed life event. Because of this methodological shortcoming we decided to develop an interview to investigate friendship difficulties over the last 12 months. We would then be in a position to evaluate the role of ongoing friendship difficulties in the presence and absence of recent life events collected over the same time. Furthermore we wished to measure friendships rather than peer relations in general because the present evidence suggests that psychological as well as social factors will determine who is regarded as a 'friend'. For example, it is apparent that children discriminate between friends and peer relations on the basis of how well they know another child (Berndt, 1984; Rubinstein, 1984). In addition, the meaning of 'friendship' for children involves emotional investment as well as a consideration of the availability and adequacy of the social relationship (Hartup, 1983); friends are likely to arise only if children have sufficient opportunity to meet other children (Berndt, 1983).

We have reported findings concerning the method and measurement of the quality of children's friendships (Goodyer *et al*, 1989). These findings indicated that puberty represented a point of change in the effects of friendships difficulties. Thus, while pre-pubertal children were overall equally likely to be anxious or depressed, post-pubertal children were significantly more likely to be anxious in the presence of moderate to poor friendships. There were however no significant differences in the proportion of boys or girls experiencing friendship difficulties and sex exerted no effects on the three-way interaction between moderate to poor friendships, puberty, and diagnosis. The findings reported in that research raise many questions concerning the meaning and the mechanisms of friendship difficulties that require further clarification. For example, how should the psychological impact of poor friendships be considered in the presence of other variables such as recent stressful life events? Both these adversities may exert independent effects on the risk of developing emotional disorder. Alternatively, they may be related such that the effects of one type of adversity are dependent on the presence of the other.

This paper reports data testing three main hypotheses:

(a) that friendship difficulties and recent stressful life events exert independent effects on the risk of developing emotional disorder in school-age children
(b) that emotional disorder in children between the ages of 7 and 16 is significantly more likely in the presence of a moderate to severely undesirable recent life event than in the presence of moderate to poor friendships
(c) that the three-way interaction between moderate to poor friendships, puberty, and diagnosis is altered in the presence of recent stressful life events.

Method

A cross-sectional case-control study was carried out in the City of Salford. Psychiatric cases were obtained from the clinics of the University Department of Child and Adolescent Psychiatry, based in a paediatric hospital containing out-patient psychiatric services for children and adolescents, and a psychiatric hospital containing out-patient and in-patient services for adolescents up to 18 years of age. Control subjects were obtained from general practice case registers in the same urban area.

Selection of the cases

All patients attending the two clinics were considered potential research subjects. Inclusion criteria for the study were:

(a) age between 7 and 16 years
(b) no mental handicap
(c) living with mothers or mother surrogates for at least two years before referral
(d) mothers or mother surrogates free of recent psychoses or serious medical conditions likely to impair their ability to take part in research interview
(e) suffering a disorder whose onset could be identified as occurring within the last 14 months, so that retrospective recall of life events was no longer than 26 months
(f) met criteria for emotional disorder described below.

Onset of disorder was defined as the appearance of new symptoms or a marked and identifiable change in the frequency, duration and quality of signs and symptoms, which resulted in referral. The 100 clinical cases can be classified in three groups in terms of onset of disorder:

(a) 38 with no evidence of signs or symptoms before onset
(b) 32 with reported vague distress which did not affect the child's life, who developed definable new signs and symptoms resulting in referral; onset was taken at the point of new symptoms being reported
(c) 30 with reported vague distress whose degree, frequency and quality had altered and resulted in referral; these emergent symptoms resulted in referral because previously they had not been causing difficulties for the child or altering his/her everyday living but were now doing so. Onset was taken at the point where symptoms were reported as emergent.

Criteria for emotional disorder

Mothers (or their surrogates) completed the Rutter A(2) parent questionnaire on their first visit to the clinic. Those children who were definitely conduct disordered on the basis of their scores were excluded. Those children with definite emotional or mixed disorders and those whose scores were ambiguous (i.e. too low and therefore uninterpretable) were subject to a clinical screen, consisting of mental state examination of the child and interview with the mother in all cases and family interviews in half.

One of the authors (IG) then established with the clinicians whether the child was suffering from an emotional disorder. This was defined as a two-week history (minimum) of predominant features of altered mood characterised by anxiety, fearfulness, sadness or misery, accompanied by one or more of the following features: phobic or obsessional symptoms, sleep disturbance, eating disturbance, alimentary symptoms, or social withdrawal (including school refusal).

Children whose clinical picture contained these features but dominated by behavioural characteristics such as lying, stealing, firesetting or destructiveness were excluded. In the ten cases where clinicians remained in doubt, children were reinterviewed by IG and a consensus diagnosis agreed. Of these ten cases four were included in the study.

Finally, all cases were subject to two further classifications:

(a) *anxiety-dominant* disorders, presenting with marked features of fearfulness, phobias, obsessions, and somatic symptoms (headache, abdominal pain and other non-specific pains); features of misery and sadness were transient or absent

(b) *depressive-dominant* disorders, presenting with marked and persistent features of misery and sadness accompanied by one or more negative thoughts (hopelessness, helplessness, suicidal ideation, poverty of thought), and one or more somatic features (undesirable alteration in sleep, appetite, energy, performance); anxiety symptoms may also be present but are not predominant clinical features. Therefore no concurrent diagnosis of anxiety was made.

All cases reported here were collected between August 1984 and October 1985. Of 1100 children referred to the services participating in the study, 121 met the criteria for inclusion, and 115 (95%) agreed to participate. Thus new-onset emotional disorders in school-age children represented approximately 10% of all referrals.

Criteria for community controls

Community controls consisted of children identified from the case registers of two general practices in the health district: practice 1, an inner-city general practice which houses part of the University Department of General Practice and serves a predominantly (> 80%) working-class population as identified by their own case register using the Registrar General's classification (classes 3 (manual), 4, 5); and practice 2, a suburban general practice associated with the university whose population is approximately 50% middle-class (classes 3 (non-manual), 2, 1): where parents were unemployed for over two years the family was identified as social class 6. The age, sex, and social class of each case was identified at the time of interview.

The social class of the child was used to determine which general practice case register a control would be selected from. For each case a control subject was identified of the same sex and age to within six months (three months older or younger) whose surname began with the same letter. A second, reserve control was also identified.

When a control had been selected from the register he/she was subject to a brief screening procedure to exclude medical and psychiatric disorder. The children's health records were examined by the general practitioners, who vetoed cases if they had treated them for emotional/behavioural difficulties. The mothers of the control subjects underwent a preliminary interview. If the child had been seen by a school doctor or psychologist for emotional/behavioural difficulties in the previous month or if such difficulties were present at interview they were excluded. Twelve were excluded.

Eighty of the first controls were used. Ten families had moved and were untraceable, two were in hospital for medical treatment, and eight refused to enter the study. Thus 20 reserves were used. Of the 100 controls 60 were selected from practice 1, and 40 from practice 2. Of the 20 controls not used from the first-control sample, 14 were from practice 1 and 6 from practice 2. The first 100 cases were matched with 100 controls for age and sex and group matched for social class.

The results presented in this paper are based on the analysis of this matched group ($n = 200$).

Measurement of friendship

Firstly, we were concerned to measure the effects of recent friendship rather than attempt to make inferences about longer-term relationships spanning years and different developmental stages. We therefore confined ourselves to inquiry about the last 12 months. Secondly, we were concerned to measure the everyday organisation of friendships and not a particular set of circumstances that might arise. Therefore we established that we were not identifying patterns of friendships occurring at a time of substantial crisis such as a family death, or as a consequence of a unique experience resulting in unusual notoriety, such as gaining admirers through recent personal achievement. We therefore took into account components of social network, frequency, duration and number of contacts with children, the social setting in which contacts occurred, the reciprocity of contacts, and the preference and affiliation of contacts.

Children presenting with long-term continuous difficulties in socialisation were excluded because they would not meet the entry criteria for recent-onset emotional disorder described below. Some children, however, may well have had episodes of friendship difficulties in the past and these were not measured in the present interview.

A semistructured interview schedule was developed which obtained substantive information about the ongoing nature of friendships. A description of the child's friendship was then constructed on the basis of all available information derived from the interview, including background information collected concurrently from the mother. A check on potential independence was carried out at this stage by IG and CW using a consensual rating, so that only information considered certainly or almost certainly independent of disorder was included. The description of the child's friendships used for rating consists of the confluence of child's and mother's contextual information. Unresolvable disagreement between the mother and child occurred only twice, both psychiatric cases and both 15-year-old girls.

Friendship was rated on a three-point scale (good, moderate, or poor) by consensus between IG and CW.

The length of recall time for cases and controls was different: for controls it was the last 12 months, whereas for cases it was to 12 months before onset, and as the duration of disorder was 1–12 months (mean 8.6 months, s.d. 5.1 months), recall time for cases was 13–24 months.

Full details of the methods and measurements of friendships, including data indicating satisfactory reliability for test–retest of mother–child pairs, reports of friendships and the rating of friendships by objective raters, are given in a separate paper (Goodyer *et al*, 1989).

Recent stressful life events

Recent life events were measured using the child and adolescent Life Event Schedule (2nd version). The interviewer inquires by a means of a general paragraph about a given class of event (accidents, illnesses, marriages, bereavements, legal, natural disasters) and then specifically about each event in that class. Detailed questioning determined the exact nature and circumstances of each event that had occurred in the 12 months before onset of disorder for the cases or day of interview for the controls.

The schedule was administered to the mothers of all the subjects, and more than 90% of the interviews took place in the child's home. One of the authors (CW) was responsible for over 85% of the interviews.

The likelihood that a life event was independent of illness was rated on a five-point scale ranging from 'not independent' to 'almost certainly independent' (Brown & Harris, 1978). The degree to which an event would exert an undesirable effect on the child likely to last days or weeks was also rated on a five-point scale ranging from 'not undesirable' to 'almost certainly undesirable'. This rating is derived from the negative-impact rating reported by Paykel (1983) and is based on the concept of long-term threat developed by Brown & Harris (1978). All events received both ratings which, were obtained by consensus between IG and and CW.

Only events that were almost certainly independent of illness and carried a moderate to severe degree of undesirability were used in the analysis. Previous research has suggested that mild events are not significantly associated with emotional disorder (Goodyer *et al*, 1985). Some reliability data have been previously published (Paykel, 1983; Goodyer *et al*, 1985).

The identification of puberty

We were not able to subject children to exact puberty measurement. Therefore a comprehensive interview inquiring about the physical changes associated with puberty was used. Both mothers and children were interviewed.

The interview was based on Tanner's (1962) criteria for pubertal development. Puberty was considered present when any one of three criteria were present in the previous 12 months (Marshall & Tanner, 1969, 1970):

(a) a sustained growth spurt reported by either child or parent
(b) a sustained increase in size of testes or breast development
(c) the development of other secondary sexual characteristics.

Therefore we endeavoured to establish retrospectively the presence or absence of puberty over the same time frame as the inquiry about friendships and life events rather than at the point of interview.

Statistics

Comparisons were made between cases and controls for the proportion of subjects experiencing moderate to poor friendships and one or more moderate to severely undesirable life events. Sex and puberty were included in the analysis.

Logistic regression (GLIM program) was used for examining independent and interactive effects of variables (Fleiss *et al*, 1986). This technique determines the best fit of the data and calculates the log odds or probability of being a case. The statistic reported is the scaled deviance, which approximates the χ^2 when the fitted model is correct. Further details of the method and tabulated logistic-regression data are available on request.

Results

The characteristics of the sample

There were 51 girls and 49 boys in each group. The mean duration of symptoms in the psychiatric group was 8.6 months (s.d. 5.1). The age range of the cases was 7–16 (mean 12.7, s.d. 2.6). The age range of the controls was the same as for the cases (mean 13.1 years, s.d. 2.5). There were no significant differences for age between the two groups ($t = 1.0$). The cases consisted of 67% working-class and 33% middle-class subjects and the controls were 55% working-class and 45% middle-class subjects ($\chi^2 = 3.0$, NS).

Of the 100 cases 32 were classified as depressed and 68 as anxious (thus in school-age children a diagnosis of anxious-dominant disorder is twice as likely than depressive-dominant disorder in new-onset cases presenting with emotional symptoms). The prevalence of new-onset emotional disorders at a child and adolescent psychiatry service is approximately 3% for depression and 6% for anxiety.

The influence of social class

The procedure for matching social-class groups used in the study may lead to a bias in the proportion of subjects reporting social adversity despite the lack of significant differences overall. Comparisons were therefore made between cases and controls for the effects of social class on life events and friendship difficulties. Of 122 working-class children, 66 (54%) experienced one or more moderately to severely undesirable life events compared with 36 of 78 middle-class children (46%) ($\chi^2 = 1.50$, NS). Of the 122 working-class children, 42 (34%) experienced friendship difficulties compared with 22 of the 78 middle-class children (28%) ($\chi^2 = 0.84$, NS).

The influence of age

We have previously demonstrated that there are no significant age differences in the proportion of cases or controls experiencing one or more recent undesirable life events (Goodyer *et al*, 1987). These results were replicated in this study, where 38 of 71 (53%) cases aged over 12 experienced one or more events compared with 17 of 31 of controls (53%) ($\chi^2 = 0.01$, NS). Similarly, 28 of 48 cases (58%) reporting moderate to poor friendships were aged over 12 compared with 10 of 16 controls (63%) ($\chi^2 = 0.08$, NS). Therefore no significant age effects are present in the proportion of children experiencing life events or friendship difficulties.

Friendships, events, sex, and puberty

Table I shows the distribution of cases and controls experiencing one or more recent moderate to severely undesirable life events and moderate to poor friendships. Seventy-one cases reported one or more events compared with 31 controls ($\chi^2 = 32.0$, d.f. $= 1$, $P < 0.001$); 48 cases reported moderate to poor friendships compared with 16 controls ($\chi^2 = 23.5$, d.f. $= 1$, $P < 0.001$). The combination of the two recent adversities is considerably more common in cases than controls (33% v. 6% respectively) than either adversity is when it occurs alone (38% v. 25%, 15% v. 10% for events and friendships, respectively) (Table I).

The findings suggest that the likelihood of being a case rather than control may be best explained by considering events and friendship difficulties together. The potential interaction between the two factors however cannot be adequately investigated from a χ^2 analysis. For these purposes a logistic regression is required so that the independent and interactive effects of events and friendships can be determined. In addition the effects of both sex and puberty need to be investigated to determine whether the associations between the social adversities and the probability of emotional disorder is influenced by these personal attributes.

Table II shows the associations between friendships, events, sex, and puberty. The best fit was a model in which the two factors life events and friendships were found to have an additive interaction on the log of the odds in favour of being a case rather than a control. This additive model was not significantly improved when the effects of sex and puberty were taken into account (see footnote to Table II). These results suggest that these two recent adversities exert independent effects on the probability of being a case or control.

TABLE I

Numbers of cases and controls experiencing adversity

	Cases (n = *100*)		*Controls* (n = *100*)
	anxious (n = *68*)	*depressed* (n = *32*)	
No moderate/severe events or moderate/poor friendships	9	5	59
Moderate/severe life events only	25	13	25
Moderate/poor friendships only	11	4	10
Both moderate/severe events and moderate/poor friendship	23	10	6

The results show that exposure to either of these two recent adversities results in a similar magnitude of risk for the likelihood of developing emotional disorder: moderate to poor friendships increase the relative odds by a factor of 4.9; moderate to severely undesirable life events increase the relative odds by a factor of 5.5.

From Table I we have ascertained that in a third of cases both adversities occurred compared with some 6% of controls. Therefore we need to establish the magnitude of risk likely to occur when severe events and friendship difficulties occur in combination. The additive interaction between the two recent adversities results in increasing the relative odds of being a case by a factor of (4.9×5.5) 26.95 (the additive effect of the two independent effects is obtained by a multiplication of their known odds).

An indication of how moderate/poor friendships and one or more moderate/severe life events relate to each other can be obtained from further analyses of the data in Table II. The analyses for cases and controls were undertaken independently as relationships between the same social factors may be different for different groups of children (Goodyer *et al*, 1988).

For cases, a model fitted in which life events were independent of friends, sex, and puberty. By comparison, friends and sex were shown to be conditionally independent given puberty (scaled deviance $= 5.794$, d.f. $= 9$, NS). In other words, in order to understand the origins and effects of friendship difficulties, a knowledge of puberty is required. In order to understand the origins and effects of life events, however, no knowledge of puberty or sex is required. The conditional relationships between puberty and sex does not involve either of the social adversities measured in the study and is therefore not relevant to the investigation of the origins or effects of these adverse factors.

A similar analysis was carried out for controls (again using the raw data shown in Table II). In this analysis a model fitted in which all four factors were independent (scaled deviance $= 6.2744$, d.f. $= 11$, NS).

Thus life events and friendships are unrelated to each other in both cases and controls. This suggests that the origins (as well as effects) of these two recent adversities are independent of each other.

Diagnosis

It may be that the presence of both moderate to severely undesirable life events and moderate to poor friendships has an effect on the form of disorder, particularly if puberty and sex are entered into the analysis.

Table I shows the proportion of anxious and depressed cases reporting neither, one, or both social adversities. The proportions of anxious and depressed children in each cell are equivalent. This suggests no significant difference between anxious and depressed cases for the type or combination of events and friendship difficulties.

Table III shows diagnosis and social adversities cross-tabulated with sex and pubertal status. Fourteen of the 32 depressed children (44%) were boys and 18 (56%) were girls. Nine boys (65%) and five girls (28%) were pre-pubertal, and five boys (35%) and 13 girls (72%) were post-pubertal

TABLE IIA
Friendship and life-event status by pubertal status and sex

	Cases				*Controls*			
	Boys (n=49)		*Girls (n=51)*		*Boys (n=49)*		*Girls (n=51)*	
	Pre-pubertal (n=32)	*Post-pubertal (n=17)*	*Pre-pubertal (n=22)*	*Post-pubertal (n=29)*	*Pre-pubertal (n=27)*	*Post pubertal (n=22)*	*Pre-pubertal (n=25)*	*Post-pubertal (n=26)*
Good friends								
Events ($n=63$)	16	4	8	10	5	5	6	9
No events ($n=73$)	6	2	4	2	19	13	14	13
Moderate/poor friendships								
Events ($n=39$)	8	8	8	9	1	1	3	1
No events ($n=25$)	2	3	2	8	2	3	2	3

GLIM computation fits a model that the probability of being a case or control depends only on friends and events status and not on sex or puberty.

(a) Scaled deviance = 7.509 at cycle 4, d.f. = 11, NS (with sex and puberty):

Estimate	*s.e.*	*Parameter*
2.177	0.4589	1
−1.645	0.3764	Friend (2)
−1.704	0.3306	Events (2)
−0.1321	0.3336	Sex (2)
−0.2711	0.3375	Puberty (2)

(scale parameter taken as 1).

(b) Removing sex and puberty. Scaled deviance = 8.452 (change = 0.943) at cycle 4, d.f. = 13, NS (change = +2) (without sex and puberty):

Estimate	*s.e.*	*Parameter*
1.929	0.3643	1
−1.580	0.3667	Friend (2)
−1.697	0.3288	Events (2)

(scale parameter taken as 1).

(Scaled deviance approximates χ^2 when the model is a good fit.)

TABLE IIB
Logistic regression model of factors predicting emotional disorder

Friendships	*Adverse life events*	*Sex*	*Puberty*	*No. of cases observed (n=100)*	*Total no. of children (n=200)*	*Fitted*	*Residual*
Moderate/poor	No	Boys	Pre	2	4	2.231	−0.233
Moderate/poor	No	Boys	Post	3	6	3.347	−0.285
Moderate/poor	No	Girls	Pre	2	4	2.231	−0.233
Moderate/poor	No	Girls	Post	8	11	6.136	1.132
Moderate/poor	Yes	Boys	Pre	8	9	7.859	0.142
Moderate/poor	Yes	Boys	Post	8	9	7.859	0.142
Moderate/poor	Yes	Girls	Pre	8	11	9.605	−1.454
Moderate/poor	Yes	Girls	Post	9	10	8.732	0.255
Good	No	Boys	Pre	6	25	5.362	0.309
Good	No	Boys	Post	2	15	3.093	−0.698
Good	No	Girls	Pre	4	18	3.506	0.296
Good	No	Girls	Post	2	15	3.093	−0.698
Good	No	Boys	Pre	16	21	12.315	1.633
Good	Yes	Boys	Post	4	9	5.278	−0.865
Good	Yes	Boys	Post	8	14	8.210	−0.114
Good	Yes	Girls	Post	10	19	11.142	−0.532

TABLE IIIA

Friendship and life-event status by pubertal status, sex and diagnosis

	Depressed (n = 32)				*Anxious (n = 68)*			
	Boys (n = 14)		*Girls (n = 18)*		*Boys (n = 35)*		*Girls (n = 33)*	
	Pre-pubertal	*Post-pubertal*	*Pre-pubertal*	*Post-pubertal*	*Pre-pubertal*	*Post pubertal*	*Pre-pubertal*	*Post-pubertal*
Good friends								
Events	3 (33%)	2 (40%)	2 (40%)	6 (40%)	13 (57%)	2 (17%)	6 (35%)	4 (24%)
No events	2 (22%)	1 (20%)	0	2 (15%)	4 (17%)	1 (8%)	4 (24%)	0
Moderate/poor friendships								
Events	4 (45%)	1 (20%)	2 (40%)	3 (20%)	4 (17%)	7 (58%)	6 (35%)	6 (38%)
No events	0	1 (20%)	1 (20%)	2 (15%)	2 (9%)	2 (17%)	1 (16%)	6 (38%)

GLIM computation fits a model that the probability of being anxious or depressed depends only on the interaction friends × puberty and not on sex or life events.

(a) Scaled deviance = 18.356 at cycle 3, d.f. = 15, NS.
Scaled deviance = 8.931 at cycle 4, d.f. = 10, NS.

Estimate	*s.e.*	*Parameter*
−0.6833	0.5333	1
−0.7257	0.6381	Friends (2)
−0.5066	0.6506	Puberty (2)
0.09360	0.4658	Sex (2)
0.08503	0.5059	Events (2)
2.287	0.9240	Friends (2) × puberty (2)

(scale parameter taken as 1).

(b) Scaled deviance = 9.002 at cycle 3, d.f. = 12, NS.

Estimate	*s.e.*	*Parameter*
−0.6190	0.4682	1
−0.7309	0.6316	Friends (2)
−0.4796	0.6398	Puberty (2)
2.281	0.9069	Friends (2) × puberty (2)

(scale parameter taken as 1).

TABLE IIIB

Logistic regression model of factors predicting depressive–dominant disorder

Friendships	*Adverse life events*	*Sex*	*Puberty*	*No. of cases observed (n = 32)*	*Total no. of emotionally disordered children (n = 100)*	*Fitted*	*Residual*
Poor	No	Boys	Pre	0	2	0.700	−1.038
Poor	No	Boys	Post	1	3	0.750	−0.333
Poor	No	Girls	Pre	1	2	0.700	0.445
Poor	No	Girls	Post	2	8	2.000	−0.000
Poor	Yes	Boys	Pre	4	8	2.800	0.889
Poor	Yes	Boys	Post	1	8	2.000	−0.817
Poor	Yes	Girls	Pre	2	8	2.800	−0.593
Poor	Yes	Girls	Post	3	9	2.250	0.577
Good	No	Boys	Pre	2	6	1.235	0.772
Good	No	Boys	Post	1	2	1.222	−0.322
Good	No	Girls	Pre	0	4	0.824	−1.018
Good	No	Girls	Post	2	2	1.222	1.128
Good	Yes	Boys	Pre	3	16	3.294	−0.182
Good	Yes	Boys	Post	2	4	2.444	−0.456
Good	Yes	Girls	Pre	2	8	1.647	0.309
Good	Yes	Girls	Post	6	10	6.111	0.072

($\chi^2 = 2.14$ with Yates' correction, d.f. = 1, NS). The trend suggests that a larger sample may confirm the indication that, in new-onset cases of depression attending clinic, boys appear more likely to be depressed when pre-pubertal and girls when post-pubertal.

Thirty-five of the 68 anxious children (51%) were boys and 3 (49%) were girls. Twenty-three boys (68%) and 17 girls (51%) were pre-pubertal and 12 boys (32%) and 16 girls (49%) were post-pubertal. There are no significant differences or indications that sex or puberty influences the proportions of new-onset anxious cases attending clinic ($\chi^2 = 1.4$, d.f. = 1, NS).

We have previously demonstrated however that a three-way interaction exists between puberty, moderate to poor friendships and diagnosis, suggesting that moderate to poor friendships were significantly associated with both anxious and depressive–dominant disorders in pre-pubertal cases, but significantly more likely to be associated with anxious-dominant disorders in post-pubertal cases (Goodyer *et al*, 1988). Because of these findings the interaction term (puberty × friendships) was entered into the analysis together with sex. The purpose was to investigate whether or not including life events in the analysis would alter this three-way interaction. Sex was also included because we could not refute the possibility that this factor may exert effects in a new model involving more than one social adversity. The logistic regression showed that the best fit was the original model (footnote to Table III): the inclusion of life events does not alter the form of association between friends, puberty, and diagnosis. The absence of a sex effect confirms previous analyses.

Discussion

The results indicate that both moderate to poor friendships and moderate to severely undesirable recent life events exert independent effects on the probability of being either anxious or depressed in school-age children and adolescents. They confirm that more than one type of recent adversity exerts potentially causal effects on the probability of developing emotional disorder in school-age children. Therefore our first hypothesis that recent undesirable life events and friendship difficulties are independent in their effects is supported.

There was an approximately fivefold increase in the risk being a case rather than a control in the presence of either factor. Thus our second hypothesis that life events would exert a greater risk for emotional disorder than friendship difficulties was not supported.

Taking these two findings together is interesting, because they suggest that recent undesirable life events and friendship difficulties exert a similar degree of risk for the onset of emotional disorders but may arise from different social pathways and perhaps operate through different psychological mechanisms.

The two social adversities occurred in combination in 33% of cases. The logistic analysis indicated however that there is no significant interaction between the two variables, and their effects are additive. This of course represents a substantial risk and an important psychological interaction between two social adversities.

The fact that 6% of controls experience both adversities is of some interest. This raises the question of which other social factors (e.g. the presence of social success) or personal attributes (e.g. the presence of an easy, flexible temperamental style) protect some children from emotional disorder in the presence of significant recent social adversity.

Interestingly, the odds of being a case in the presence of life events or friendship deficits were not significantly changed by the inclusion of sex and puberty in the analysis. This does not imply that the mechanism by which boys and girls appraise such stresses are the same. There is a substantial literature indicating that there are sex differences in the mechanisms by which children develop social relations (e.g. Higgins *et al*, 1983). What the results indicate is that both pre- and post-pubertal boys and girls are exposed to the same qualities of social adversity together with the costs and benefits these experiences may bring.

The independence of life events from friendships indicates that there is no mechanism operating between these two adversities which may explain the presence of one in the presence of the other. The origins of recent undesirable events and friendship difficulties may however arise from a third factor common to both. For example, deficits in pre-school social development, mother–infant relations, or intrinsic qualities in the child such as a difficult temperament may all increase the likelihood that either of these acute social adversities may arise. It is equally the case however that the origins of these recent adversities may be substantially independent.

The inter-relationship between social factors occurring at different points in a child's life, personal attributes such as temperament or level of intelligence, and the mechanisms through which they exert their effects have only recently begun to be investigated (e.g. Masten *et al*, 1988; Goodyer *et al*, 1988). The results in this paper indicate that the three-way interaction between friendship difficulties, puberty, and diagnosis is not altered by the inclusion of recent undesirable life events in the analysis. Thus our third hypothesis that life events would alter this relationship is unsupported. Previous reports have suggested that family-focused adversities, including life events, maternal distress, and mothers reporting a lack of intimate confiding relations in their own lives, do not discriminate between anxious and depressed cases (Goodyer *et al*, 1988). These findings continue to support the notion that factors other than recent adversities (either extrinsic or intrinsic) influence the form of emotional disorder in school-age children. The inclusion of both past social adversities and personal attributes in future research may help to determine how the symptoms and form of psychopathology arises. Clearly, the significance of friendship difficulties and pubertal status will need to be taken into account in any such study. As we have seen, puberty exerts a robust and important interaction with friendships and diagnosis which is uninfluenced by life events. Indeed the association between emotional disorder and life events is uninfluenced by either sex or puberty, confirming our previous findings (Goodyer *et al*, 1987).

We cannot however determine from this research whether there are significant differences in the personal meaning of undesirable life events and friendships difficulties. The general inference from these results is that friendship difficulties are appraised differently by pre- and post-pubertal children, whereas life events are not. A finer-grain analysis of the qualities of both these adversities is required if we are to explore mechanisms in more detail. In addition, inclusion of sex-role behaviour rather than categorical classification into male and female may improve our understanding of the potential effects of gender (Wilson & Cairns, 1988). These findings do however provide further support for the view that puberty rather than age alone is an important variable in developmental psychopathology (Rutter, 1986).

Some methodological points require comment. Firstly, we have attempted to minimise potentially confounding associations between life events and friendship difficulties by the use of systematic interviewing and careful selection of cases. Nevertheless, a cross-sectional study of this type remains open to biases as a result of retrospective inquiries on a prospective population. A firmer test of the potential relationships between stressful life events and friendship difficulties would be to conduct a longitudinal community study. Under these conditions children's social experiences can be evaluated both before and after the development of disorder. Secondly, the additional use of structured interviews to classify psychiatric disorders may help to improve the specificity of findings in relation to diagnosis. A number of interviews have now demonstrated adequate reliability and discriminant validity for clinical and epidemiological purposes (Gutterman *et al*, 1987). Thirdly, the conclusions drawn from studies such as this are affected by the measures and the statistics used (Parry & Shapiro, 1986). For example, friendship measurement can be obtained from direct observation as well as interview, and a comparison of such techniques may help clarify the optimal method for a given research question. Drawing causal inferences and assigning psychological meaning to social variables depends on independent replication of findings rather than a particular statistical analysis (Brown & Harris, 1986; Rutter, 1986). Overall, however, we believe that our findings suggest different pathways for different social adversities likely to cause anxiety or depression in 7–16-year-old children.

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Childhood Sexual Experiences with Adults Reported by Women with Eating Disorders: An Extended Series

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A total of 158 women presenting with clinical eating disorders have been investigated using a self-report questionnaire and subsequent interview concerning their recollections of sexual experiences with adults before the age of 16 and later adverse sexual experiences. About a third reported events in childhood and over half described some adverse experiences. It may be that these experiences are relevant to the subsequent illness in some cases, but greater certainty must await further research. In the meantime, inquiring about such matters would seem to be advisable in the assessment and therapy of eating-disordered patients.

Anorexia nervosa and bulimia remain disorders of uncertain cause. Nevertheless, psychological and social factors are generally thought to be important in their pathogenesis. Of these factors, the individual's response to biological maturity and the psychological and social demands of sexual development are widely invoked as being especially relevant (Crisp, 1980, 1984; Garfinkel & Garner, 1982). Events which disrupt that development, or make premature demands upon the person, might be expected to increase the risk of eating disorder. Child sexual abuse is now recognised as being not uncommon (Finkelhor, 1979, 1984, 1986; Nash & West, 1985). The later consequences of such experience are certainly varied and seem to be usually disadvantageous. They include problems with self-esteem, sexual confidence, and personal relationships (Browne & Finkelhor, 1986). An association between childhood sexual abuse and later eating disorder is therefore plausible.

Clinical case descriptions of anorexia nervosa have sometimes made mention of adverse sexual experiences, including incest (e.g. Crisp, 1984). However, recently the possibility of an association has been discussed and investigated directly. Sloan & Leichner (1986) reported that five out of six patients in an anorexia nervosa treatment programme gave histories of sexual abuse in childhood. This observation led them to inquire more systematically, and 9 out of 23 subsequent patients gave a history of "past or present sexual abuse or incest".

Finn *et al* (1986) studied a sample of subjects attending "women's therapy groups in three types of settings", namely a women's therapy collective, a women's centre specialising in the therapy of sexual abuse, and groups held by private-practice clinicians. However, of 140 women approached, only 87 (62%) co-operated. A self-report screening questionnaire was used to detect possibly abused women, who were later interviewed, and possible eating disorder was detected by the self-report Eating Patterns Questionnaire. Seventy per cent of respondents reported "sexual abuse", but there was no association with reports of "eating disorder and subclinical eating disturbance", which were likewise common in the study subjects. The authors suggested that the high rates of both sexual abuse and eating disturbance "may have led clinicians to perceive a relationship where none exists". However, the study failed to define sexual abuse adequately, and also did not differentiate adverse sexual experiences in adulthood from childhood abuse. Furthermore, eating disorder and 'disturbance' were diagnosed by self-report, and clinical diagnoses were not made, although 16 subjects gave responses suggestive of current bulimia. Also, the settings studied may have resulted in the selection of subjects especially likely to report abusive experiences, whether or not they also had eating problems. Thus the hypothesised link between childhood sexual abuse and adult eating disorder of clinical severity is not refuted by this study.

Calam & Slade (1989) carried out a not dissimilar questionnaire study on 130 female university students, unselected for morbidity or presentation for therapy. The response rate was 70%. They used the Eating Attitudes Test (EAT) and a Sexual Events Questionnaire (SEQ). Overall, 60% of respondents reported "some form of unwanted sexual experience", although definition is unclear. Positive associations were found between scores on the two instruments, although again this study did not directly address the relationship between clinically diagnosed eating disorder and defined childhood sexual abuse.

Calam & Slade (unpublished conference proceedings) also hypothesised that intrafamilial abuse would be associated with low-weight anorexia nervosa rather than normal-weight bulimia because self-starvation could serve to "punish the parent who has exploited or failed to protect her". Their results included positive association between the dieting subscale of the EAT and intrafamilial sexual experience, but no such association was found with the bulimia subscale. This provided some support for their hypothesis.

The present authors have previously published rates of defined childhood sexual abuse reported at special investigation by 78 clinically diagnosed eating-disordered subjects (Oppenheimer *et al*, 1985). We now report an extension of that series, with further analyses including examination of the above hypothesis of Calam & Slade.

Method

The subjects were consecutive female referrals to an eating-disorder clinic who fulfilled DSM–III criteria for either anorexia nervosa or bulimia (American Psychiatric Association, 1980). The investigation of the history of childhood sexual abuse was incorporated as a routine part of the initial assessment. The patient usually attended with a relative or other informant and would be interviewed by the psychiatrist while her companion was interviewed by the social worker. The investigation of childhood sexual experiences was carried out on all patients within the period of initial assessment.

The investigation itself had two parts and was conducted by the social worker (RO) or sometimes by a female junior doctor. Firstly, each patient was invited to complete a self-report questionnaire, known as the Sexual Life Events Inventory (SLE), adapted from Finkelhor (1979). The inventory invites respondents to describe in the terms of the questionnaire any sexual experiences before the age of 13 with someone over 16; and then from ages 13 to 15 inclusive with someone five or more years older. Such definitions are clearly arbitrary but do exclude experiences with peers, and most of the events reported in fact involved much older people. However, for completeness, the questionnaire also asked subjects in a third section to describe *any* unpleasant or coercive sexual experience which they may have had. (Such events are referred to as 'non-criterion experiences'.) The questionnaire invites respondents to describe up to three events, or series of events, in each age category. It records details of the nature of the event, the characteristics of the other person involved, and matters such as the use of force or threat and the subject's own response. The categories of events are listed in Table I. When more than one sexual activity occurred with one perpetrator, or more than one event occurred within a category, the activity recorded was the most 'serious' according to the hierarchy set out on Table I. (This hierarchy should not be taken as necessarily implying degree of possible effect upon the subject.)

The second part of the investigation was an unstructured interview with the social worker in which the respondent could further discuss her responses to the questionnaire. The investigation itself was often the beginning of therapeutic discussion of the issues raised.

The questionnaires were subsequently coded and analysed with the aid of a computer.

Results

Of the 158 subjects included in the study, 80 had anorexia nervosa and 78 had bulimia. A further two patients were approached but refused to co-operate with this part of the clinical assessment. One had seemed ambivalent about attendance in general and appeared affronted by the nature of the inquiry. She refused to complete a questionnaire and dropped out of contact with the clinic. The other became very distressed when confronted by the questionnaire but remained in touch. In the course of subsequent therapy it emerged that she had been seriously sexually abused as a child. However, she did not complete the investigation in the same manner as the other subjects and her case has not been included in the series.

All subjects were over the age of 15 (mean age 24 ± 6.3, range 15–49 years). Only three subjects were under 16 at the time of investigation and all were over 15½. Sixty-one subjects were social class I and II, 25 were class III non-manual, 33 were class III manual, and the remainder were from social classes IV or V or were unclassified (by father's occupation using the Registrar General's classification).

Forty-nine subjects (31.0%) reported experiences within the criteria of childhood abuse outlined above and a further 42 (26.6%) reported events only within the extended criteria. Sixty-seven (42.4%) reported no events within either set of criteria. Over 80% of the events reported involved 'contact abuse'. Table I gives the details of the most 'serious' event for each subject. The other person involved was male in all but two cases, and was known to the subject in 80% of cases. There was no significant association between the type of eating disorder and the rate of abuse nor age category of abuse (Table II). The types of event reported were varied. For the subjects with events before the age of 13, there were two reports of full sexual intercourse. In the 13–15 age group, there were 15 reports of full intercourse (Table I). No definite cases of buggery were reported. The questionnaire does not make specific reference to anal intercourse but the issue was attended to within the subsequent interview where appropriate. In at least one case the subject was uncertain which orifice had been penetrated.

In the younger age group, the other person was on average 27.3 years (s.d. 15.8, range 7–80 years) older than the subject, and was a family member in half of the cases (including seven fathers, one stepfather, and three grandfathers). The event was described as having been initiated by the other in 97% of cases, as involving some actual force in 41%, some threat in 38%, and severe or moderate distress at the time in 75%. Only one subject reported any pleasure.

In the older age group (13–15 years inclusive), the other person involved was on average 17 years older (s.d. 12.8, range 5–46 years), and was a family member in a third of cases (including five fathers and one stepfather). The event

TABLE I

Reported events with childhood categories arranged according to a hierarchy of 'seriousness' of the sexual act

	Subject aged under 13 years	*Subject aged 13–15 years*
Full sexual intercourse	2 (6.3%)	15 (57.7%)
Subject having oral contact with genitals of others	1 (3.1%)	0
Subject receiving oral contact to genitals	2 (6.3%)	0
Intercourse without penetration	4 (12.5%)	2 (7.7%)
Subject touching other's genitals	5 (15.6%)	1 (3.8%)
Other touching subject's genitals	3 (9.4%)	1 (3.8%)
Subject fondling other sexually	0	0
Other fondling subject sexually	6 (18.8%)	2 (7.7%)
Subject exposing own genitals	1 (3.1%)	0
Witnessing abusive sexual acts between others	1 (3.1%)	0
Other exposing genitals to subject	5 (15.6%)	1 (3.8%)
Kissing and hugging sexually	0	3 (11.5%)
Request to do something sexual	2 (6.3%)	1 (3.8%)
Total	32	26

Within each age category an individual is included only once and is listed under the most 'serious' event category. However, an individual may appear in both age categories if she reported events in both and if the other person involved was different.

TABLE II

Subjects reporting events within each category by diagnosis

	Event when under 13	*Event when 13–15*	*Non-criterion event*	*No event*	*Total*
Anorexia nervosa	13	11	20	36	80
Bulimia	19	6	22	31	78
Totals	32	17	42	67	158

Each subject is classified only once according to the earliest category in which she reported an event.

was described as having been initiated solely or mainly by the other in 74% of cases, as involving force in 39%, threat in 35%, and severe or moderate distress in 66.6% of cases. In this age group some pleasure was associated with 22% of events.

TABLE III

Age at first event and family/non-family relationship with perpetrator examined by diagnostic group

	Anorexia nervosa		*Bulimia*	
	Under 13[1]	*13–15*	*Under 13*	*13–15*
Family	6	8	11	5
Non-family	6	3	8	1

1. One subject was omitted because the relationship with the other person was unclear.

Model examining Calam & Slade hypothesis: $\chi^2 = 0.05$, d.f. = 1, NS.

For those reporting criterion events there was no significant association between diagnosis and whether the incident was intrafamilial (incestuous) or extrafamilial. Table III examines this issue and the age of the subject at the time of the event. Log-linear modelling (Bishop *et al*, 1975) was used to examine possible interactions between these variables but none was statistically significant.

As mentioned above, subjects were invited to describe, in addition to the criterion childhood events, any other upsetting or unpleasant sexual events, whether in childhood or when adults. Fifty-six subjects reported such 'non-criterion events', of whom 41 had not reported criterion events in childhood. Many of these reports were of sexual encounters as adults and included 25 instances of full sexual intercourse, in 21 of which there was some threat and in 19 some actual force. Such events were often construed as rape. Childhood events reported were those in which the other person involved was too young to meet the criterion, for example repeated forcible digital penetration of an 11-year-old girl by her 14-year-old brother. Altogether these non-criterion events involved strangers in 11.5% of cases and included only three cases of incestuous contact. Most of the others involved were described as 'known persons' or as 'friends'.

Thirteen subjects reported more than one event or sets of events. To count as separate 'events' the other person involved must have been different. Fifteen subjects reported both criterion and non-criterion events.

Discussion

This study reports surprisingly high rates of defined adverse sexual experiences in women presenting with eating disorders. About a third of the subjects reported sexual experiences in childhood with others who were significantly older. The average age difference between the people involved was substantial. Furthermore, the types of events described were by no means trivial, even when the subject was aged under 13. The majority of the events were experienced as distressing at the time and could plausibly be suspected of carrying a risk of lasting effects upon the individual. A number of our subjects had experienced more than one abusive relationship. Whether multiple experiences reflect the summation of chance events, or are the result of individual vulnerability based upon psychological or social factors, is unclear and will remain so until suitable comparisons with control data can be made.

Important questions remain concerning the specificity and causal relevance of these findings. A number of studies have suggested that a substantial proportion of psychiatric patients in general may report childhood sexual abuse. Rosenfeld (1979) in an early but small study reported that 6 out of 18 female psychotherapy patients proved to have had incestuous experiences. Carmen *et al* (1984) conducted a survey of the case notes of 188 psychiatric patients, both male and female, and recorded notations of both physical and sexual abuse: 'sexual abuse' was reported by 27% of the females but this was not clearly defined and seemed to include adult experiences. More recently, Jacobson & Richardson (1987) interviewed 50 female and 50 male psychiatric patients, seeking information on their lifetime experience of 'assault'. Again, the definition of assault was unclear but 22% of the women and 16% of the men gave some account of 'sexual abuse' in childhood. Current research in our own centre also suggests that a substantial proportion of non-eating-disordered female psychiatric patients give histories of childhood sexual abuse. It seems unlikely, therefore, that such histories are specifically associated with anorexia nervosa and bulimia. This issue cannot be resolved until adequate samples of different diagnostic groups have been studied using the same method, including, crucially, the same definitions of abuse.

Similar problems of method and definition arise in trying to address the issue of whether or not the apparently high rates of sexual abuse reported in the present study are in excess of those that would be expected in the general population. Such an excess would be necessary if a hypothesis of a causal role for these experiences in later disorder is to be sustained. An adequate comparison group would need to be representative of the general population from which the psychiatric sample was taken and, furthermore, it would need to be studied using the same method of investigation and the same definitions of abuse. No such comparison has yet been conducted. However, such data as are available suggest that childhood sexual abuse, broadly defined, is reported by non-psychiatric populations in broadly similar proportions to those reported here.

Nash & West (1985) in their studies of 223 women aged 20–39 drawn from a general practice register, and of 92 female students, found overall rates of 'abuse' of 42% and 54% respectively. However, their definition included at least one category, namely receipt of obscene phone calls, that has not usually been included in other studies. Finkelhor (1979, 1984) in his studies of 530 female students and 334 mothers in the USA used similar categories to the present study and produced estimates of childhood abuse of 19% and 15% respectively. Clearly, these four estimates on different populations may vary because the true rates vary or because of differences in sampling, method, or definition. The last of these factors is especially important in appraising the literature but it can be partly overcome by comparing rates of an activity for which there should be good agreement about definition, namely full sexual intercourse with penetration. In all, 17 of our subjects (10.8%) reported full intercourse with adults before the age of 16. Nash & West (1985) reported rates of full sexual intercourse of 3.2% in their student sample (3/92) and of 1.8% in their general practice sample (4/223), although they used slightly wider age categories. The rate for this kind of sexual event is markedly higher in our sample and the possibility that this could have causal relevance in some cases cannot be dismissed.

A community study in New Zealand (Mullen *et al*, 1988) provides important evidence to support the idea that rates of psychiatric disorder in general may be higher in women giving a history of childhood sexual abuse and hence for the possibility of a causal role for such experiences.

The present study can do little more than raise the question as to whether childhood sexual contact with adults may in some cases play a part in causing later clinical eating disorder. Such a suggestion is plausible and the effects of these experiences upon self-esteem and sexual confidence may well be the mediating variable. Such effects may have acted as 'setting conditions' (Slade, 1982) which rendered the individual vulnerable to later disorder. The hypothesis is compatible with the evidence so far available, although the link is unlikely to be specific. Comparative data on rates both in other psychiatric groups and in general populations are as yet inadequate. More confident statements must await further research.

In the meantime, it would seem wise to incorporate some attention to the issue of adverse sexual experiences into the assessment and therapy of patients with anorexia nervosa and bulimia. Our experience suggests that the vast majority of such patients tolerate inquiry into such matters and many find it helpful, even if initial disclosure is sometimes distressing. We hold the view that these reports are to be construed as true memories rather than fantasies. The accounts are convincing and we feel that it is appropriate that they should be believed unless there are special reasons to suggest otherwise. In some of our cases independent corroboration was available, including admission of the events by the adult involved. Many abused subjects seem relieved to be given an opportunity to talk about their experiences, often for the first time. To have their relevation met with disbelief could be damaging. Conversely to have serious attention paid to these often personally important events may be a good start to continuing wider therapy.

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The Influence of Social Factors on Common Mental Disorders
Destabilisation and Restitution

DAVID GOLDBERG, KEITH BRIDGES, DIANE COOK, BARBARA EVANS and DAVID GRAYSON

This study distinguishes between processes that cause individuals to experience symptoms – destabilisation – and those that are associated with loss of symptoms over time – restitution. It is shown that different clinical, social, and personality variables are associated with each of these processes. Where destabilisation is concerned, it is shown that different variables were associated with the development of symptoms of anxiety and those of depression. Different variables were associated with restitution, and they did not show the same relationship with the symptom dimensions of anxiety and depression as those which were associated with destabilisation.

Surveys carried out in community settings invariably reveal a substantial proportion of patients with psychological disorders which fall short of criteria required for a formal psychiatric diagnosis. These patients are described as 'false positives' on screening questionnaires and, if they are labelled at all, their psychological distress is referred to by terms such as 'transient situational disturbances' or 'adjustment disorders' (Goldberg & Huxley, 1980). When such patients are followed up systematically, the majority will be found to have remitted spontaneously (Goldberg & Blackwell, 1970; Brodaty, 1983; Grayson *et al*, 1987, 1990). Indeed, it can be argued that in order to satisfy criteria for 'caseness', which require symptoms to have lasted for at least two weeks (American Psychiatric Association, 1987), it is necessary for two conditions to be satisfied: the individual must have begun to experience symptoms, and processes of normal homeostasis must have failed. We call the former process *destabilisation*, while the latter is referred to as a failure of *restitution*. Individuals with transient disorders can be thought of as those who have destabilised but are able to restitute spontaneously. It seems likely that different social and environmental variables are associated with each of these processes.

The aim of the present study is to investigate the social and personality characteristics which co-vary with destabilisation and restitution. The former can be accomplished by studying those environmental variables that are related to possession of symptoms at the inception of an episode of illness, including in the study all those seeking medical care whether or not they are psychologically distressed. We therefore document those environmental variables that are related to symptom experience when help is first sought. The latter can be accomplished by discovering which variables are related to symptom loss over time, and confining the analyses to those patients receiving research diagnoses of mental illness.

Our earlier research (Goldberg *et al*, 1987) has indicated that the symptoms that are commonly encountered in primary-care settings can be conceptualised in two-dimensional space, representing the dimensions of anxiety-related and depression-related symptoms. Any individual can be given a location in this two-dimensional space, depending upon the particular combination of symptoms reported at psychiatric interview. Although conventional clinical wisdom separates anxiety states from depressive disorders, it is certain that the two symptom dimensions are highly correlated in community settings, and it seems of interest to investigate whether they can be shown to have different determinants.

A secondary aim of the study is therefore to discover whether the social variables studied exert their effects along a single, general dimension of mixed anxiety/depression, or whether it can be shown that different social variables exert their effects on the separate symptom dimensions. In analysing such a complex data set (bivariate dependent variables of anxiety and depression, and multiple independent variables), one must be aware of the possibility of spurious associations, or of 'proxying' by one variable of another. For instance, it may be that sex influences anxiety symptom counts. Yet, if substantial correlation exists between anxiety and depression, there will also be an association between sex and depression. If this latter association is fully accounted for by the anxiety–depression correlation, it may be spurious to say sex also affects depression. Similarly, one variable can act as a proxy for another as a potential cause. For example, if sex affects anxiety and if sex is correlated with age, then age will be 'associated' with anxiety spuriously. In both these examples, univariate analyses are incapable of teasing apart such relationships and of protecting against the emergence of indirect or spurious associations.

Our intention is to explore the potential causes of destabilisation and restitution by first segregating the possible causes into subgroups homogeneous in their effects in bivariate illness space, and then performing multiple-regression analyses of illness within each subgroup to tease apart genuine effects from spurious ones. The exploratory nature of this research should be emphasised for two reasons. First, the putative causes were measured by retrospective self-report at initial consultation simultaneously with the symptom counts, so that causal inferences concerning destabilisation are based only on correlational data. Second, we analyse simultaneously many variables on 191 subjects. We have therefore been conservative in reducing the data where statistically indicated.

The present analyses are a secondary analysis on a data set which was collected in order to investigate the factors that distinguish somatic forms of psychiatric disorder from entirely psychological forms of illness. As Hyman (1972) has pointed out, such secondary analyses can have several limitations. Those that apply to the present study are that the illnesses seen are not a random sample of illnesses in primary care, for reasons explained below. The independent variables used in this study were those thought to be of interest for the original aim of the study, and therefore do not include several otherwise interesting variables which might well have improved the results reported below had they been included.

Method

The sample consisted of 191 patients complaining of new episodes of illness in 15 general practices in the Greater Manchester area. The sample is less than that reported in companion papers (Goldberg *et al*, 1987; Goldberg & Bridges, 1987), since in order to be included it was necessary that a full social assessment was available by an independent researcher (BE) using the Social Interview Schedule (SIS; Clare & Cairns, 1978). The social interview was carried out with an independent informant in the patient's home within two weeks of the initial consultation. The patients were interviewed by one of us (KB) using the Psychiatric Assessment Schedule (PAS; Dean *et al*, 1983) with the addition of questions dealing with the nature of the patient's present complaint and its likely aetiology, as well as the Social Stress and Support Interview (SSSI; Jenkins *et al*, 1981). In addition, we had available ratings made by the general practitioner (GP) concerning the patient's complaint and its presumed aetiology.

The sample of 191 patients was not random, since those presumed psychologically well (on the basis of the GP's rating *and* a low score on the General Health Questionnaire (GHQ)) were undersampled relative to those presumed ill (high GHQ score *or* thought psychologically ill by GP) in the ratio of 1 : 2.7. For the studies dealing with the determinants of destabilisation, the patients who had been undersampled were 'weighted back' to represent a notional 264 patients with consecutive new illnesses for whom social assessments were available. However, in order that tests of significance would be based on an appropriate number of degrees of freedom, this weighted data set was then reduced back to a total of 191 patients, so that the number of 'notional patients' (who now *do* represent a random sample of inceptions of illness) was now equal to the number of actual patients seen on the survey.

The *dependent variables* are depression and anxiety symptom counts derived from the PAS. Each subject is assigned a score out of 10 for anxiety, and a score out of 14 for depression, using scales derived from our earlier latent-trait analysis (Goldberg *et al*, 1987: see Table 2 of that paper for the prevalence of each symptom in this setting, and Table 4 for the symptoms loading on the latent traits for anxiety and depression). The scores of subjects on these two scales correlate with one another +0.63.

The potential causes of destabilisation and restitution are explored by first segregating the possible causes into subgroups homogeneous in their effects in bivariate illness space, and second performing multiple-regression analyses for each of these subgroups separately, in order to tease apart genuine effects from spurious ones. The *independent variables* derived from the two social assessments (SIS and SSSI) were reduced to 0/1 dichotomies wherever possible by using median splits, and are shown in Table I, together with the values for level 1 of each dichotomy. Thus, for example, 'occupational stress: stressed' means that this item of the SSSI has been scored in such a way that the half of the subjects with the most stress at work have been assigned a score of unity, and the remainder assigned a score of zero. Had variables been left continuous it would have raised issues about the appropriateness of scaling in building quantitative regressions later on. By dichotomising we are simply opposing subjects unambiguously low and high on the construct in question. This is more sensible in an exploratory analysis.

Strategy for the destabilisation analysis

The first stage of analysis consisted of taking each of the variables in Table I and performing a multivariate t-test using initial anxiety and depression symptom counts as the dependent variables. Such an analysis can be thought of as a t-test which first asks: is there a significant difference between the two groups of subjects (0,1 on the Table I variable) in bivariate symptom space? It also produces the 'direction' in bivariate space along which the two groups most differ. This direction is given as a univariate linear combination of the depression and anxiety score. For instance, we might find that sex (1 = female, 0 = male) has a maximum effect in the direction:

$$y = 0.36\ (\text{anxiety score}) - 0.04\ (\text{depression score})$$

The 'loadings' +0.36 on anxiety and −0.04 on depression indicate that the effect of female sex is essentially directly on anxiety (there will, however be a male/female depression difference, but only of the magnitude expected by the correlation between anxiety and depression of +0.63).

Having performed such analyses on each putative cause, we attended further only to those with a P value <0.10. This rather liberal rate was used because we wish at this stage only to select a small subset of variables for the final regression stage of analysis, where the more conventional criterion of $P<0.05$ is adopted.

The second stage takes this reduced set of independent variables and classifies them into one of four groups, depending on their anxiety and depression loadings. These groups are A (loading mainly on anxiety, for example, sex in the above example), D (loading mainly on depression), A+D (loading nearly equally on anxiety and depression), and A−D (loading on both, but opposite in sign). In this way all the putative causes in any one such group have their maximal effect in essentially the same direction of bivariate illness space.

If the load on one dimension was more than twice that on the other, the variable was assigned to that dimension. Variables that failed to satisfy this condition had either same-sign loadings or different-sign loadings on both dimensions. In the former case they were assigned to A+D, in the latter to A−D. This strategy enabled variables to be assigned to the nearest of the four dimensions in which they exerted their maximal variation.

Within each group, we finally conducted multiple-regression analyses, with the univariate dependent variable defining that group (anxiety symptom count, depression symptom count, total symptom count or anxiety−depression symptom difference, respectively). In this way we examined the independent contributions to morbidity of potential causes acting in homogeneous directions in bivariate illness space, while controlling for confounding effects among these causes. For example, suppose sex and work status individually show an association with anxiety, but that the latter's association is a spurious result of its correlation with sex – perhaps women are more likely to be unemployed. If this were so, the partial-regression coefficient for work status would be zero (or non-significant) when sex is included in the multiple-regression equation, while that for sex would be non-zero, indicating a genuine contribution of sex to variation in anxiety.

The prior step of gathering together variables roughly homogeneous in their expression in bivariate illness space protects us from another form of proxying of one variable by another. Variables mainly associated with depression will also *appear* to be associated with anxiety, because of the correlation between the two dimensions. In allocating each variable to its prime direction, we have ensured that no variable can simultaneously appear as a possible cause of both depression and anxiety, although we allow variables to contribute as possible causes of both dimensions.

This overall analysis strategy acknowledges that common forms of psychiatric disorder are not univariate in their manifestations, and allows us to explore the possibility that there are distinct causes for variation along the two major dimensions of symptoms.

Strategy for the restitution analysis

The 130 patients who were found to be psychiatrically unwell by the research psychiatrist at the inception of their illness were followed up and interviewed in their homes using the same clinical assessments six months after their initial consultation with the family doctor. The analysis of the follow-up data was identical to the earlier analyses, with two exceptions: first, no weighting procedure was used, since all 130 patients who were followed up had been assigned a DSM-III psychiatric diagnosis at initial interview; second, the dependent variables were anxiety and depression *symptom losses* over the six months following initial consultation with the family doctor. The correlation between these two measures was +0.43.

Results

Destabilisation

Table I presents the results of the multivariate discriminant analysis (bivariate t-tests), with anxiety and depression symptom counts at initial consultation being the dependent variables. The Eigen value reflects the ratio of between-group: within-group variance in the maximal direction of effect. Loadings on anxiety and depression are reported only for those putative causes with a P value less than 0.10. These loadings reflect the direction in symptom space in which particular variables maximally act, and should not be interpreted as measures of size of the effect. The final column denotes the group into which the causal variable was placed for multiple regression (and, thus, the dependent variable for that regression). The most salient feature of Table I is that putative causes seemed to move subjects into either anxiety or depression regions of illness, rather than general illness (A+D). It is not surprising that only one variable defines groups differing in the (A−D) direction. To do so one group of subjects would simultaneously need to be higher on anxiety and lower on depression than the other, and in the destabilisation analysis most variables are opposing predominantly psychiatrically well subjects with those who are ill one way or another.

Table II summarises the results of the multiple-regression modelling. If we consider that for depression, then 12 variables from Table I with significant loads on depression were put into an initial regression. We had initially intended to define interaction variables to explore synergistic or antagonistic relationships between the putative causes: however, with 12 main effects there would be 66 possible *two-way* interactions alone. A data set derived from only 191 subjects could not possibly carry a model so rich in parameters with any reliability. We have therefore attempted to 'flag' social variables of potential importance for future research in what we see as an exploratory analysis, rather than attempt to build an explicit quantitative model involving higher-order interactions in accounting for scale-dependent variance.

Of the 12 main effects, eight had non-significant ($P>0.05$) partial-regression coefficients. A second regression was therefore performed with the remaining four putative causes.

A test was performed comparing initial and second models, and showed that the omitted eight variables removed no variance in depression beyond error. The second regression was final, as each variable had a significant partial-regression coefficient. This procedure was adopted in all analyses below (including the restitution phase), and only the final models are reported.

The β coefficients in Table II indicate, for example, where 'trait anxiety' is concerned, the difference between the two groups (e.g. high or low on trait anxiety) in units of the dependent variable (i.e. number of symptoms), with all other variables in the model held constant. Thus, subjects with a high score on trait anxiety have 1.16 more depression symptoms than those with low scores, when they have the same amount of support from family and confidant and when they are equally satisfied with their interaction with relatives. Similarly, subjects who are simultaneously high on trait anxiety, dissatisfied with relatives' interaction, and report little support from family or confidant have $(1.16+1.49+0.75+0.67)$ 4.07 more depression symptoms than those simultaneously at the other levels of these four variables. It can be seen that lack of close support increases depression, and the amount of missing support seems related (those with low support from both family and confidant have more depression than either alone). All statistical tests in all regressions were performed at the $P=0.05$ level. The R^2 value of 0.30 for depression indicates that 30% of the variance in depression was explained by the model reported in Table II.

Anxiety seems related to an assessment by the psychiatrist that the aetiology of the patient's problem is entirely psychological, or that there is a mixture of psychological and organic factors at work. It can also be seen that higher levels of anxiety symptoms occur with those who have had problems for longer periods of time before their initial consultation.

The influence of a high vocabulary score and having symptoms other than autonomic or pain appears to protect against a general psychiatric malaise, measured by (A + D) in this model: however, it can be seen that the magnitude of this effect (reflected in the value of R^2) is relatively slight compared with factors that affect anxiety or depression separately.

The (A − D) variable of opportunities for interaction with relatives is omitted from Table II. When such opportunities were reported as restricted, subjects' A − D scores were 0.83 less than when not so restricted ($P=0.01$, $R^2=0.03$). That is, such restrictions lead to a relative increase in depression and drop in anxiety symptoms.

The R^2 of 0.81 for the A regression seems very high. One possible reason why it may be spuriously high is that the putative causal factors are confounded by symptom state, since both aetiological assessments and symptom scores are obtained simultaneously. Similarly, the variables dealing with 'duration of illness' include as 'duration 0' all those not suffering a physical (or psychiatric) illness at all. Thus, such variables are not independent of current symptom state. The same may be said of trait anxiety ('Do you often feel tense?' etc.).

Table II therefore also presents additional regression analyses where any variables which might be confounded by current symptom state were omitted from consideration. In comparing these results with those already reported, the most salient feature is the emergence of variables relating to stress and unemployment that previously were 'overshadowed' by the variables which have now been removed. The model which accounts for 31% of the variance in anxiety is therefore more conservative, and probably more appropriate. Such 'overshadowing' is precisely what would be expected were these removed variables restatements of symptom state, since genuine causes could account for little additional symptom variance while other (proxy) measures of symptoms had been unknowingly included as independent variables in the model. We have therefore removed the same variables from the analyses to be reported later in our section dealing with restitution.

For depression, however, the omission of trait anxiety did not allow other variables to emerge, so it is probably sound to stay with the original model, and to regard trait anxiety as a safe indicator of predisposition to depressive illness. Similar arguments apply to the (A + D) model.

Briefly then, lack of support from a 'close other' and trait disposition are associated with the onset of depressive symptoms, whereas stress particularly related to work and income are associated with anxiety, even when sex is controlled for (as in a multiple-regression model). In addition, there is a sex effect on anxiety (females more anxious) above and beyond that induced by sex differences in work and income status. With these views we have accounted for about 30% of variance in each of the dependent variables. Note that we could account for about a further 10–15% of the variance in depression if we included the anxiety variables in the depression model, by relying on the correlation between anxiety and depression of 0.63. If, for example, being morbidly anxious led to being depressed, then it would be appropriate to include such causes as indirect causes of depression. However, in the present work we have made no such assumptions.

Restitution

This part of the research examines the determinants of *symptom loss*, whether or not such determinants are clearly causal. For example, being an 'owner occupier' seems unlikely to have of itself a causal influence on restitution, but it may act as a proxy for some causal process, perhaps the possession of a wide range of social advantages.

We thought it wise to omit the duration of illness variables in order to avoid circularity (long illnesses last a long time, and vice versa), and we included two new variables: 'mode of presentation' assessed by the psychiatrist (somatisers, psychologisers, and a mixed group), and 'type of complaint' as assessed by the GP (entirely physical, entirely psychiatric, and a mixed group). The first stage of the analysis is shown as Table III. The most salient feature is that 7 of the 13 predictors exerted their influence in the A − D direction, while only four appeared to lead to recovery in either anxiety or depression alone.

Table IV shows the final regression models. The factors predicting loss of depression symptoms (and of anxiety, but no more than that related to the correlation of 0.43 in

TABLE I

Inception of illness: effects of individual variables in bivariate symptom space for 191 subjects

Dichotomous variable	*High value indicates*	*Eigen value*	P *value*	*Anxiety loading*	*Depression loading*	*Scale for regression*
Sociodemographic						
Age	older	0.01	0.29			
Sex	female	0.07	0.00	0.36	−0.04	A
Marital status	with spouse	0.02	0.20			
Employment status	[1]	0.15	0.00	0.36	−0.03	A
Aspects of presentation						
Pain symptoms	present	0.01	0.47			
Autonomic symptoms	present	0.09	0.00	0.27	0.10	A
Other symptoms	present	0.06	0.00	0.20	0.19	A+D
Psychiatric aetiology	present	0.49	0.00	0.42	−0.04	A
Organic aetiology	present	1.15	0.00	0.46	0.03	A
Psychiatric/organic aetiology	present	0.23	0.00	0.34	0.03	A
Duration (physical)	long	1.36	0.00	0.47	0.04	A
Duration (psychiatric)	long	3.85	0.00	0.61	0.15	A
Personality variables						
Educational level	high	0.02	0.21			
Vocabulary score	high	0.03	0.06	0.13	0.25	A+D
Social desirability	high	0.02	0.18			
Defensiveness	high	0.01	0.57			
Whitely index	high	0.06	0.01	0.40	−0.15	A
Locus of control	high	0.00	0.83			
Trait anxiety	high	0.14	0.00	0.09	0.31	D
Social Stress and Support Inventory (from patient)						
Occupation: stress	stressed	0.04	0.03	0.29	0.07	A
Occupation: support	supported	0.01	0.54			
Money: stress	stressed	0.01	0.25			
Money: support	supported	0.01	0.40			
Housing: stress	stressed	0.04	0.02	−0.02	0.37	D
Housing: support	supported	0.02	0.21			
Family: stress	stressed	0.00	0.88			
Family: support	supported	0.03	0.09	0.03	0.34	D
Confidant: stress	stressed	0.08	0.00	0.12	0.27	D
Confidant: support	supported	0.12	0.00	0.13	0.27	D
Social Interview Schedule variables (from independent information)						
Accommodation	poor	0.00	0.84			
House, ?owned	not owned	0.00	0.73			
Residential stability	unstable	0.00	0.69			
Household care	poor	0.01	0.39			
Satisfied with house	dissatisfied	0.03	0.10	0.07	0.31	D
Household management	difficulties	0.01	0.25			
Household composition	inadequate	0.00	0.88			
Income: amount	inadequate	0.10	0.00	0.28	0.09	A
Income: management	difficulties	0.00	0.77			
Income: satisfaction	dissatisfied	0.00	0.83			
Leisure: opportunity	restricted	0.01	0.33			
Leisure: extent	inadequate	0.02	0.15			
Leisure: satisfaction	dissatisfied	0.09	0.00	−0.03	0.39	D
Social contacts: extent	inadequate	0.06	0.01	−0.19	0.46	D
Social contacts: satisfaction	dissatisfied	0.10	0.00	0.01	0.37	D

(Continued)

TABLE I *(Continued)*

Dichotomous variable	*High value indicates*	*Eigen value*	P *value*	*Anxiety loading*	*Depression loading*	*Scale for regression*
Interactions with neighbours						
opportunities	restricted	0.01	0.41			
quality	difficulties	0.02	0.10			
satisfaction	dissatisfied	0.11	0.00	0.02	0.36	D
Interactions with relatives						
opportunities	restricted	0.04	0.04	−0.41	0.50	A−D
quality	difficulties	0.05	0.01	−0.15	0.50	D
satisfaction	dissatisfied	0.15	0.00	0.04	0.38	D

1. Six types of employment status were examined, which could not be dichotomised on *a priori* grounds. Following the multivariate *t*-tests, these were collapsed into three groups represented by the dichotomies shown in Table II: full time or retired (1) versus other four (0); and unemployed or student (1) versus other four (0).

The loadings in columns 5 and 6 show only the *direction* of each effect with respect to anxiety and depression, not the size or nature of the effect.

TABLE II

Inception of illness: multiple-regression models indicating relative contributions of individual variables to the causation of anxiety and depression respectively, and to 'general severity' of dysphoria

	Beta	*Significance*
Anxiety (no additional effect on depression)		
Multiple-regression analysis		
long duration psychological symptoms*	4.07	0.00
entirely psychological aetiology*	0.78	0.03
both organic and psychological aetiology*	0.72	0.05
long duration physical symptoms*	−1.32	0.10
	$R^2=0.81$	$P<0.000$
Additional multiple-regression analysis (omitting variables* which might have been reflecting current illness)		
full-time work or retired	−1.73	0.001
unemployed or not earning	−1.63	0.024
adequacy of income	1.57	0.001
female sex	0.84	0.001
stress at work	1.33	0.002
stress in social life	1.46	0.001
	$R^2=0.31$	$P<0.000$
Depression (no additional effect on anxiety)		
Multiple-regression analysis		
interaction with relatives–dissatisfied	1.49	0.01
high trait anxiety*	1.16	0.002
supported by family	−0.75	0.03
supported by confidant	−0.67	0.02
	$R^2=0.30$	$P<0.000$
Additional multiple-regression analysis (omitting variables* possibly reflecting current illness)		
interaction with relatives–dissatisfied	2.50	0.000
supported by confidant	−1.03	0.000
supported by family	−0.76	0.031
	$R^2=0.21$	$P<0.000$
General severity (depression and anxiety)		
Multiple-regression analysis		
other symptoms present*	−2.41	0.003
vocabulary score	−1.36	0.08
	$R^2=0.08$	$P<0.001$
Additional multiple-regression analysis (omitting variables* possibly reflecting current illness)		
vocabulary score	−1.84	0.02
	$R^2=0.03$	$P<0.02$

TABLE III

Symptom loss over six months: effects of individual variables in bivariate symptom space for 130 subjects

Dichotomous variable	*High value indicates*	*Eigen value*	P *value*	*Anxiety loading*	*Depression loading*	*Scale for regression*
Sociodemographic						
Age	older	0.01	0.58			
Sex	female	0.01	0.73			
Marital status	with spouse	0.00	0.85			
Employment status	[1]	0.04	0.82			
Aspects of presentation						
Pain symptoms	present	0.03	0.12			
Autonomic symptoms	present	0.01	0.54			
Other symptoms	present	0.01	0.57			
Psychiatric aetiology	present	0.05	0.04	0.38	−0.21	A − D
Organic aetiology	present	0.04	0.10	0.31	0.07	A
Psychiatric/organic aetiology	present	0.01	0.42			
Mode of presentation	[1]	0.09	0.03	−0.15	0.30	A − D
Type of complaint	[1]	0.12	0.01	−0.05	0.29	D
Personality variables						
Educational level	high	0.01	0.67			
Vocabulary score	high	0.02	0.24			
Social desirability	high	0.00	0.92			
Defensiveness	high	0.04	0.09	−0.35	0.23	A − D
Whitely index	high	0.04	0.10	0.19	0.18	A + D
Locus of control						
personal	high	0.02	0.28			
chance	high	0.11	0.001	−0.07	0.30	D
others	high	0.00	0.92			
Trait anxiety	high	0.03	0.19			
Social Stress and Support Inventory (from patient)						
Occupation: stress	stressed	0.01	0.73			
Occupation: support	supported	0.03	0.20			
Money: stress	stressed	0.01	0.67			
Money: support	supported	0.03	0.12			
Housing: stress	stressed	0.01	0.73			
Housing: support	supported	0.01	0.70			
Family: stress	stressed	0.01	0.53			
Family: support	supported	0.02	0.28			
Confidant: stress	stressed	0.01	0.58			
Confidant: support	supported	0.02	0.37			
Social Interview Schedule variables (from independent information)						
Accommodation	poor	0.00	0.88			
House, ?owned	not owned	0.05	0.07	0.19	0.18	A + D
Residential stability	unstable	0.02	0.27			
Household care	poor	0.03	0.15			
Satisfied with house	dissatisfied	0.07	0.03	−0.16	0.30	A − D
Household management	difficulties	0.04	0.12			
Household composition	inadequate	0.00	0.76			
Income: amount	inadequate	0.01	0.68			
Income: management	difficulties	0.02	0.33			
Income: satisfaction	dissatisfied	0.03	0.23			
Leisure: opportunity	restricted	0.02	0.41			
Leisure: extent	inadequate	0.02	0.32			
Leisure: satisfaction	dissatisfied	0.02	0.29			

(Continued)

TABLE III *(Continued)*

Dichotomous variable	*High value indicates*	*Eigen value*	P *value*	*Anxiety loading*	*Depression loading*	*Scale for regression*
Social contacts: extent	inadequate	0.08	0.01	−0.35	0.24	A − D
Social contacts: satisfaction	dissatisfied	0.03	0.20			
Interactions with neighbours						
opportunities	restricted	0.00	0.77			
quality	difficulties	0.08	0.01	0.40	−0.17	A
satisfaction	dissatisfied	0.08	0.01	−0.21	0.30	A − D
Interactions with relatives						
opportunities	restricted	0.04	0.09	−0.24	0.30	A − D
quality	difficulties	0.02	0.25			
satisfaction	dissatisfied	0.03	0.20			

1. These variables allowed no dichotomisation on *a priori* grounds, so they were entered with all levels in the above analyses. Those variables with systematic variation in two-dimensional space were dichotomised in the way shown in Table IV.

TABLE IV

Symptom loss over six months: regression models showing factors contributing to restitution of symptoms

Multiple-regression analysis	*Beta*	*Significance*
Overall severity (depression + anxiety)		
Whether owner occupier	−2.3	0.03
Whitely index	−1.8	0.07
	$R^2 = 0.07$	$P < 0.000$
Depression (no additional effect on anxiety)		
Health locus of control − chance	−2.25	0.0003
Entirely physical presenting complaints	−2.47	0.003
Mixed presenting complaints	−2.79	0.0002
	$R^2 = 0.20$	$P < 0.000$
Anxiety (no additional effect on depression)		
Quality of interaction with neighbours	−1.5	0.03
Entirely organic aetiology	−1.4	0.03
	$R^2 = 0.09$	$P < 0.005$
Anxiety/depression imbalance (anxiety−depression)		
Satisfaction with housing	−1.83	0.005
Opportunities for interactions with relatives	−1.35	0.03
Defensiveness	1.38	0.03
Purely somatic presentation	1.29	0.05
	$R^2 = 0.19$	$P < 0.000$

symptom-loss measures) were having a low locus of control score, and being classified by the GP as having an entirely psychiatric complaint. This latter effect probably relates to illness recognition and subsequent treatment during the six-month interval. Predicting loss of anxiety symptoms, predominantly, were having reported few difficulties with neighbours and being classified as having an entirely psychiatric aetiology or organic aetiology exacerbated by psychiatric state.

Subjects' improvement was in the direction of a relatively greater loss of anxiety than depression symptoms (larger value of A − D) if they presented as purely somatising, they were more defensive, they reported satisfaction with their housing, and/or they reported adequate opportunities for interactions with their relatives. Those on the opposite poles of these dichotomies lost relatively more depression than anxiety symptoms over the follow-up period (smaller value of A − D). Conversely, those dissatisfied with their housing had a relatively greater loss of depression and/or a smaller loss of anxiety symptoms than those satisfied (their depression loss, on average, exceeded their anxiety loss by 1.83 symptoms); similarly those with restricted opportunities for interaction with relatives lost 1.35 more depression symptoms than anxiety symptoms.

The (A + D) regression was more complicated statistically. Both the Whitely index and whether the patients owned their houses were significant ($P < 0.1$) in Table III. Neither were significant alone in regressions, but in the joint regression the ownership variable achieved $P = 0.03$, while the Whitely Index had $P = 0.07$; omitting both altogether gave a

P value of 0.000. Rather than omit discussion altogether, this latter regression is reported in Table IV. Those low on the Whitely index or owning their own home show more general (A + D) psychiatric improvements (model $P = 0.000$, $R^2 = 0.07$).

The models for D and (A – D) both accounted for about 20% of variance, the others less than 10%. While these amounts are much smaller than the variances explained in the destabilisation study, it must be remembered that altogether on any one measure (say, depression) we could account for more variance.

Discussion

We have attempted to delineate some putative causal relationships among various precursors of illness and distinct symptom dimensions. The analysis is speculative and exploratory, but does tend to support a two-dimensional model, with each dimension relating to different environmental variables.

Destabilisation seems well explained in terms of variables which appear to act in a fairly distinct way on the two dimensions of anxiety and depression. Of necessity, anxiety and depression were assigned the same causal priority in this study. We did not contemplate models where, say, lack of support causes anxiety which in turn causes depression. Such sophisticated modelling is probably premature and is certainly beyond the scope of the data set we had available. Were such a situation true, then causes of anxiety are, *ipso facto*, causes of depression – yet the present analysis is still informative. For the reasons explained above, the present study is certainly an improvement on the univariate forms of analysis which seek only to describe one-illness/one-cause relationships, in no way addressing the more complex possibilities which must surely exist.

Despite strong data reduction and forcing potential causes to contribute to only one (of four) types of illness, we have accounted for at least 30% of the variance in anxiety and depression. It must be recalled that this is a secondary analysis of a data set collected for another purpose, and that the measuring instruments used (the SIS and the SSSI) are both fairly general instruments: had we included measures of variables shown in other research to be related to affective illness such as provoking life events, negative self-esteem, or measures of intimacy, it seems likely that more variance could have been accounted for (Ingham *et al*, 1987; Brown, 1990).

The results for restitution, although significant, were not as strong, with only about 20% of the variance in 'depression loss' and 'depression/anxiety imbalance' being accounted for. Subjects were found not to have moved directly towards normality, but a number of factors discriminated subjects whose recovery paths were from depression to anxiety, or vice versa. Thus, factors that correlated with restitution were not only not the same as those which related to destabilisation, but discriminated subjects in terms of an asymmetry of symptom loss. Thus, some subjects moved from a state of high depression and anxiety with a relatively greater loss of depressive symptoms. The factors that were related to a relatively greater loss of depressive symptoms were not having somatic presenting complaints; being dissatisfied with housing; not being defensive; and having restricted opportunities for interaction with relatives.

The finding that variables relating to housing and social links with relatives and neighbours are related to restitution is broadly in line with previous findings (Huxley *et al*, 1979; Johnstone & Shepley, 1986), while the finding that those having no restrictions in opportunities for interactions with relatives had relatively greater loss of anxious symptoms fits well with Brown *et al*'s (1977) finding that those with increased integration into the community (and thus, presumably, fewer restrictions for interactions with relatives) had increased anxiety and decreased depression.

Tyrer (1985) has argued that common neuroses are indivisible, and Kleinman (1988) stated that "depression and anxiety disorders resulting from social causes can be reconceived not as discrete diseases, but rather as non-specific bodily forms of human distress . . . why distinguish these problems simply because at times they exhibit distinctive forms of symptom presentation, when they share the same social origins?" A diametrically opposite point of view was put forward by Kendler *et al* (1987), who argued that while genetic factors are non-specific, environmental factors are responsible for determining whether a given individual develops symptoms of depression or anxiety.

Our results tend to support the latter of these alternatives: namely that the social origins of depressive symptoms are distinct from those of anxiety-related symptoms. We have also argued that different environmental variables predict the liability to develop symptoms in the first place, and the ability to lose symptoms once they have developed. We have also presented preliminary data about environmental factors which may be associated with a movement from a state of anxious depression to a state of anxiety.

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Violent Death and Mental Illness

A Study of a Single Catchment Area over Eight Years

ELIZABETH KING and BRIAN BARRACLOUGH

The names of 412 residents of the catchment population of a district general hospital unit who died potentially self-inflicted deaths in the eight years 1974–81 were identified. They were classified as suicide (245), accidental death (126), and undetermined (41). In each group, over half had a lifetime history of psychiatric treatment and over a third were psychiatric patients at the time of their death. The relative risk of a violent death for those who died within a year of their last psychiatric contact was 27 times greater than that of residents with no recent psychiatric contact. The relative risk was highest for those aged 35–44 and lowest for those of 75 years and over.

Mental illness is known to be associated with suicide. Many suicide verdicts include the rider "while the balance of the mind was disturbed". Retrospective studies of consecutive suicides based on interviews with relatives report that up to 94% were mentally ill at the time of their death (Robins *et al* (St Louis), 1959; Dorpat & Ripley (Seattle), 1960; Jacobson & Jacobson (Brighton), 1972; Barraclough *et al* (West Sussex), 1974; Chynoweth *et al* (Brisbane), 1980; Arato *et al* (Budapest), 1988). Other studies report increased rates of suicide in persons with a history of mental illness (Innes & Millar, 1970; Koranyi, 1977; Rorsman *et al*, 1982; Black *et al*, 1985; Martin *et al*, 1985).

An association has also been shown between mental illness and deaths from similar causes to suicide of an accidental or 'undetermined' nature (Holding & Barraclough, 1975, 1977). Increased rates of accidental and undetermined death in populations of psychiatric patients have been reported (Black *et al*, 1985; Martin *et al*, 1985).

The quantitative relation between these forms of violent and unnatural death and mental illness in a population served by a single psychiatric service has not previously been described. For this reason we studied a hospital catchment area, rather than a coroner's district, to provide an account of the forms of death in which psychiatrists have a particular interest in the population for which they are responsible.

Our objectives were: to identify all those residents of the catchment area who died violently; to compare suicides and accidental and undetermined deaths with respect to the victim's history of psychiatric illness; to compare the pattern of violent deaths in the catchment area with that in England and Wales; and to calculate the rates and relative risk of violent death for patients seen by a psychiatrist within a year of their death.

Method

The catchment area of the Royal South Hants Hospital Department of Psychiatry comprises the City of Southampton, its suburbs to the east and west, and part of the New Forest. The population size (297 000) did not vary over the eight years and was similar in its age and sex distribution to that of England and Wales.

From January 1974 until May 1979, Knowle Hospital, a traditional county mental hospital, situated 21 km east of Southampton, provided the psychiatric service for adults. In May 1979 a new district general hospital unit, sited in the centre of Southampton, started a comprehensive service for residents aged 15–65. The services for children, adolescents and the elderly remained unchanged.

From 1 January 1974 the Southampton Psychiatric Case Register collected demographic, social, and medical data on every resident of the city in contact with the psychiatric service and also recorded details of every contact by that resident. Information was not recorded for patients resident in the catchment area but with an address outside the city.

The study population comprised every resident of the catchment area who committed suicide, or died by a potentially self-inflicted method, between 1 January 1974 and 31 December 1981 (inclusive), whose death was registered in England or Wales, and persons of no fixed address whose death was registered in the catchment area. By specifying the area-of-residence code (Office of Population Censuses and Surveys (OPCS), 1974) for addresses in the catchment area, and the ICD E-codes for the external cause of death (World Health Organization, 1977), it was possible to discover the names of all those residents who died in England or Wales. (Deaths where no address was traceable are included in the vital statistics for the district in which the death was registered.) The details of the method are described by King (1983).

Violent deaths

A coroner's inquest must be held on all violent or unnatural deaths to determine the mode and cause of death. The coroner's findings after inquest, commonly known as the 'verdict', record the medical cause of death (e.g.

ex-sanguination), the antecedent external cause (e.g. cutting of the carotid artery), and the mode of death (e.g. suicide).

In this study we are interested in the verdict. A death from the external cause could have been:

(a) *self-inflicted*, leading to a *suicide* verdict
(b) *accidental* (by self or another) leading to verdict of *accident* or *misadventure*
(c) *deliberately inflicted* by another leading to verdict of *homicide* or *unlawfully killed*
(d) *open (undetermined)*, whether deliberately or accidentally inflicted.

Inquest verdicts of 'death by misadventure' are synonymous with 'accidental deaths' – there is no legal distinction.

Our definition of 'violent death with a self-inflicted component' includes all deaths from suicide and from external causes similar to suicide where the verdict was accident, misadventure, or open. We cast a wide net to ensure the inclusion of self-inflicted deaths which clinicians might regard as 'suicide' but which, because of the strict legal definition of suicide, were recorded as accidental or undetermined deaths. Mental illness at the time of a self-inflicted death may militate against a suicide verdict since the comprehension of the fatal consequences of the act may be so impaired by the illness that the victim did not necessarily have the intention of killing him/herself by that act. The standard of proof of intent to die is illustrated by the following examples.

> A 38-year-old married woman was admitted to the Department of Psychiatry after attempting to throw herself from a first-floor window. Later the same day she absconded from the ward in her night-clothes, and was seen running towards the nearby level crossing where she was knocked down while standing on the railway track. A verdict of death by misadventure was recorded.
>
> A 29-year-old Asian woman was admitted to Knowle Hospital after threatening to pour paraffin over herself and ignite it. While home on weekend leave she locked herself in the bathroom, poured paraffin over herself and ignited it. An open verdict was recorded.

Accidental and open verdicts may also be recorded on deaths resulting from self-endangering conduct associated with mental illness that is clearly not suicidal, such as drug and alcohol abuse (Holding & Barraclough, 1978).

Deaths registered in 1974–1978 under ICD–8 were recoded under ICD–9. The codes representing the accidental deaths were selected to correspond closely with suicide methods. Our definition of potential self-inflicted injury is wider than that of Adelstein & Mardon (1975), who included accidental poisonings only in their definition of 'estimated and possible suicides'. The ICD E-code 911 (death from asphyxiation, choking or suffocation by food/regurgitated food (vomit)) was included on the grounds that such deaths, particularly in the under-65 age group, are often associated with drug or alcohol abuse.

The inclusion of all violent deaths in this study renders irrelevant the questions of how many undetermined or accidental deaths were 'hidden suicides', and what effect the under-reporting of suicides in the official statistics might have on the results of the study.

Although our choice of ICD E-codes for accidental deaths was subjective, once chosen, every death registered with that code could be identified, and the results compared with the corresponding national statistics. However, six deaths were included where there was no possibility of self-infliction. All were included in the analysis to ensure comparability of the statistics with the national figures.

Coroners' notes

The districts of the coroners for the City of Southampton and Western Hampshire include the catchment area of the Department of Psychiatry. Both coroners held office throughout the study period and conducted 377 (91%) of the 412 inquests. The bodies of 17 victims found outside the catchment-area boundary were within the district of the coroner for Western Hampshire. Twenty more coroners conducted the 35 inquests on Southampton residents whose bodies were found in other parts of England and Wales. We inspected 411 of the 412 sets of inquest notes. The notes for one suicide could not be traced by the responsible coroner; this death was included using information from the medical notes and the death certificate.

The fact that both catchment-area coroners, each the other's deputy, were in post throughout the period of the inquiry avoids the need for an estimation of verdict reliability, particularly for deaths by drug poisoning.

Inquest notes contain: a description of the places where the event leading to death occurred and the victim was found; statements from interviews with relatives, friends, employers, and other relevant witnesses; the results of official inquiries into medical, hospital and criminal records; medical reports from attending doctors; the pathologist's report of the autopsy; and the results of toxicological investigations. All of the 411 inquest notes contained autopsy reports: 281 (68%) reported blood alcohol levels and 273 (66%) blood drug levels.

Case definition

Three criteria were considered: (a) admission to a psychiatric hospital; (b) referral to a psychiatrist; and (c) treatment with psychotropic medication by a general practitioner. We chose (a) and (b) as these reflected the severity of mental illness and gave documentary evidence. In-patient treatment alone was deemed too restrictive since so many patients who would have once needed admission, are now treated as out-patients.

Medical notes

The medical records of psychiatric treatment were located by checking the names of the dead against the indexes of patient names held at Knowle Hospital, the Royal South Hants Hospital, and the Southampton Psychiatric Case Register. Episodes of treatment up to 50 years before the patient's death were discovered in this way. For those treated outside Southampton we obtained notes from hospitals named in the inquest notes or in local case notes.

Although not everyone with a history of psychiatric treatment outside the catchment area may have been identified through the inquest notes, the number lost is probably small; all 210 with psychiatric records in the Southampton hospitals had their treatment mentioned in the inquest notes. A history of treatment for mental illness was mentioned in 231 cases, including 21 (9%) who were never treated locally.

In three cases, the family doctor's letter to the coroner in accidental (two cases) and undetermined (one case) deaths referred to hospital treatment for alcoholism, records for which proved untraceable. The use of false names, maiden names or cohabitee's names during the relevant admission may explain this. A history of psychiatric referral was recorded as such only if confirmed by hospital records. Excluded from the definition of a 'referred psychiatric patient' were those whose only contact with a psychiatrist was after the event leading to their death.

From the medical notes we compiled a record of previous psychiatric treatment which included: (a) date and age at first referral; (b) date of last consultation, diagnosis, and whether an in-patient or an out-patient; and (c) number of admissions to a psychiatric hospital.

Rates of violent death

Rates of violent death were calculated for three populations: England and Wales; the catchment area; and the City of Southampton. Census data from 1981 (OPCS, 1981) and OPCS (1974–80) population estimates for the remainder of the study period were used in the calculations.

Using case register data (Jennings, 1981, 1982) we calculated violent death rates for city residents whose last contact with a psychiatrist was less than a year before the event leading to their death (subsequently called 'recent' patients), for 'recent' in-patients, and for city residents who were not recent patients. The latter group includes both residents never treated by a psychiatrist and those whose last contact was more than a year prior to the event leading to their death.

In calculating time since last contact with a psychiatrist, the date of the event leading to death was used, not the date on which the death was certified (i.e. the date on the death certificate). Our interest was in the circumstances at the time of the fatal behaviour and not those at the time of death. For most cases these dates coincide, but in some, especially deaths from poisoning and burns, some time can elapse between the fatal act and death. An in-patient who deliberately drank a bottle of medicated shampoo survived 22 days.

Results

Between 1 January 1974 and 31 December 1981, 412 catchment-area residents aged 15 or more died violent or unnatural deaths on which a verdict of suicide, accidental death (from the selected causes), or undetermined death was returned. Three hundred and sixty residents (87%) died within the catchment area and 52 (13%) died outside it, similar percentages to those calculated for suicides occurring within and without a coroner's district (Jacobson & Jacobson, 1972). One person of no fixed abode (an accident victim) died in the catchment area.

Five suicide inquests involved more than one victim; three returned verdicts of double suicide and two suicides had committed murder before killing themselves. Seventeen (4%) deaths were those of hospital in-patients, on whom 13 suicide, one open, and three accident verdicts were recorded. Fourteen of the in-patient deaths (12 suicides, 1 open verdict, 1 accident) occurred in psychiatric hospitals and three (1 suicide, 2 accidents) in general hospitals. Of all suicides, 13 (5%) occurred in hospital, similar to the percentage reported in other studies (Crammer, 1984).

Comparison between the catchment area and England and Wales

Of the 412 deaths, there were 245 suicides (59%), 41 undetermined (open-verdict) deaths (10%) and 126 accidental deaths (31%). These 412 deaths represented 1.6% of all deaths (1974–81) in those aged 15 and over. The figures are similar for England and Wales: deaths from the same causes comprised 53% suicides, 18% open verdicts, and 29% selected accidents, accounting for 1.5% of all deaths among those 15 and over.

However, the catchment-area suicide rate is higher than that for England and Wales, with the high male rate accounting for the difference (Table I). The rate of undetermined death, nearly half that for England and Wales, could indicate that the two local coroners have a less strict definition of suicide than the average; in fact the sum of the suicide and undetermined rates does not eliminate the difference in the male rate, nor does the inclusion of the accidental deaths. The catchment area therefore has a higher male rate of suicide than the nation as a whole.

TABLE I

Number of violent deaths and rates[1] per 100 000 population aged over 15 in the catchment area and nationally

Verdict		*Males*			*Females*	
	n	*Catchment-area rate*	*National rate*[2]	n	*Catchment-area rate*	*National rate*[2]
Suicide	159	17.2	12.9	86	8.8	7.9
Open verdict	26	2.8	4.1	15	1.5	3.0
Accident	78	8.5	7.0	48	5.0	4.4
Total	263	28.5	24.0	149	15.3	15.3

1. Rates are means of annual rates for the eight years.
2 England and Wales (Office of Population Censuses and Surveys, 1974–80, 1982).

TABLE II

Violent death rates[1] *for Southampton city residents by inquest verdict and psychiatric patient status at time of event leading to violent death*

Verdict	*Recent psychiatric patients* n	*Rate*	*s.d.*	*Other city residents* n	*Rate*	*s.d.*	*Relative risk*[2] *(95% CI*[3]*)*
Suicide	77	276	78	114	8.8	2.0	31 (23–41)
Open verdict	10	36	51	23	1.8	1.2	20 (10–42)
Accident	34	122	73	73	5.6	1.8	21 (14–32)
Totals	121	434	137	210	16.2	2.5	26

Population figures used in calculations: average Southampton population aged 15 and over (OPCS, 1974–80, 1981) 164 910 (s.d 1500); average annual number of residents treated as in-patients or out-patients (Southampton Psychiatric Case Register) 3502 (s.d. 179); average annual number of patients treated as in-patients 1109 (s.d. 78).

1. Rates are eight-year average annual rates per 100 000 population at risk.

2. $$\text{Relative risk} = \frac{\text{Risk of dying a violent death if seen by a psychiatrist } (A)}{\text{Risk of dying a violent death if not seen by a psychiatrist } (B)}$$

A = No. of recent patients who died violent death/No. of patients seen by a psychiatrist in that year

B = No. of other residents who died a violent death/No. of patients in population not seen by a psychiatrist

3 Confidence intervals, calculated according to Gardner (1989).

Comparison of rates of violent death between recent psychiatric patients and other city residents

Using figures for the 1981 census population for Southampton and the average annual number of Southampton city residents with a contact recorded by the Southampton Psychiatric Case Register for the years 1974–81 inclusive, we calculated that the violent death rates of current and recently discharged psychiatric patients are significantly higher than those for other residents of the city, and that these recent patients have a greatly increased relative risk of violent death in the year following their last contact with a psychiatrist (Table II).

These rates are, of course, crude violent death rates with no account taken of the difference in age structure between the psychiatric patients and the other residents. Table III shows the rates and relative risks for different age groups after standardising the populations of recent patients and other residents against the 1981 census population of the city, which was very similar to that of England and Wales.

Psychiatric history and treatment

Over half (231) of the 412 violent death victims had been referred to a psychiatrist during their lifetime; over a third (37%) had been admitted to a mental hospital. Very little difference was found between the three verdict groups in respect of a lifetime referral (Table IV).

TABLE III

Violent death rates for Southampton city residents by age group and psychiatric patient status at time of event leading to violent death

Age group	*Recent psychiatric patients*[1] n[2]	rate	*City residents not in recent psychiatric contact* n[2]	*rate*	*Relative risk* *Recent patients/others (95% CI*[3]*)*
15–24	9	230	29	10.9	21 (10–45)
25–34	21	374	27	11.7	32 (18–56)
35–44	28	677	31	17.5	39 (23–64)
45–54	28	746	38	21.9	34 (21–56)
55–64	16	521	38	21.0	25 (14–45)
65–74	15	499	28	19.3	26 (14–48)
75 and over	4	88	19	20.9	4 (2–12)
Totals	121	432	210	16.3	26

1. Treated in year before event leading to death.
2. Total number of deaths in eight years.
3. Confidence intervals, calculated according to Gardner (1989).

TABLE IV

Comparison between psychiatric histories of violent death victims by verdict group

	Inquest verdict							
	Suicide		*Open verdict*		*Accidental*		*All verdicts*	
	n	%	n	%	n	%	n	%
Lifetime history of psychiatric care								
Never patient	99	40	19	46	63	50	181	44
Ever admitted	93	38	13	32	47	37	153	37
Never admitted	53	22	9	22	16	13	78	19
Total	245		41		126		412	100
Psychiatric patient status at time of event leading to death by verdict								
In care	49	33	11	50	22	35	82	35
current in-patient	12	8	1	5	1	2	14	6
current out-patient	37	25	10	45	21	33	68	29
Not in care	97	66	11	50	41	65	149	64
not current patient (previous in-patient care)	49	33	6	27	29	46	84	56
Not current patient (previous out-patient care only)	48	33	5	23	12	19	65	44
Total with history of psychiatric illness	146		22		63		231	
Length of time between last contact with psychiatrist and event leading to death								
Up to 1 year (recent patients)	93	64	15	68	40	63	148	64
More than 1 year	53	36	7	32	23	37	83	36
Total no. of patients	146		22		63		231	100

The proportion of suicide victims with a history of referral to a psychiatrist is similar to that found by Kraft & Babigian (1976) in Monroe County, New York.

Even when the stricter criterion of admission to a mental hospital is applied, there is little difference between the three verdict groups. These findings agree with other studies. Jacobson & Jacobson (1972) found that 32% of their Brighton suicides had received in-patient psychiatric care, while 33% of Holding & Barraclough's (1975) open-verdict sample had been psychiatric in-patients. However, the proportion of accident victims with a history of in-patient care is almost twice the 20% reported by Holding & Barraclough (1977).

Very little difference was found in the proportions of recent patients, or recent in-patients, in the three verdict groups. Two-thirds of those with a history of psychiatric illness were recent patients (Table IV), and more than half of these recent patients were current patients at the time of the event leading to their death.

The higher proportion of accidental death victims treated as in-patients is the most striking difference between the groups of psychiatric patients.

The average number of admissions, for those who had been admitted, to mental hospitals for the accidental death victims ($n = 47$, mean 4.7, s.e. 0.74) was also significantly higher than for the suicide ($n = 93$, mean 3.2, s.e. 0.35) and open-verdict ($n = 13$, mean 2.5, s.e. 0.48) patients.

Other similarities in the characteristics of the victims in the three verdict groups were found. The male : female ratio in each group was approximately 2 : 1, similar to that found in populations of suicides; the male : female ratio for those with a psychiatric history was about 3 : 2 and for non-patients was nearer to 3 : 1. Similar sex ratios for victims with and without a history of psychiatric treatment have been reported by Kraft & Babigian (1976) and by Sundquist-Stensman (1987). Their corresponding sex ratios for psychiatric patients with a history of admission was approximately 1 : 1 in each verdict group.

There was no significant difference in the proportion of suicide verdicts returned on the 231 victims with a history of psychiatric illness (63% of suicide verdicts) and the 181 without (54% of suicide verdicts).

Comparison between history of psychiatric illness among violent death victims and the general population

In each of the study years, contacts with the psychiatric service were recorded by the Southampton Psychiatric Case Register for some 2% of the population (Jennings, 1981, 1982), a similar proportion to that reported by the Camberwell Case Register (Wing & Hailey, 1972) and the Case Register in Monroe County (Kraft & Babigian, 1976). About a third (0.66%) of these were treated as in-patients.

The results of this study show that over a third of Southampton City residents who died violent deaths had been in contact with the psychiatric service in the year before their death, and over half of these patients had been admitted in that year. The pattern was similar for the catchment area as a whole, in that there was no significant difference between the proportions of victims of violent death who had a history of psychiatric referral (56% city, 58% catchment area) or were recent patients (36% city, 38% catchment area).

Discussion

The method to identify the names of people dying unnatural deaths relies on the centralised vital statistics of the OPCS. This approach avoids uncertain and time-consuming liaison with local coroners and ensures the identification of all dead residents provided their bodies were within the jurisdiction of a coroner in England and Wales. No method exists for identifying all residents whose bodies remain outside England and Wales. The number of these appears to be very small indeed and unlikely to affect our results.

Identifying dead residents with histories of contact with the Southampton psychiatric service relied on matching deceased names against the hospital's patient index. Error could arise because of omission of names from the index or the use of assumed names. The size of this error is unknown but not thought to be large. Identifying residents with histories of contact with psychiatric services beyond Southampton relied on the coroner's notes of inquest naming a hospital. The size of error from this source is again likely to be small. This is because all the dead with histories of psychiatric contact in Southampton, a fact checked independently of the coroner's notes, had this fact in the coroner's notes. This suggests that the coroner's inquiry systematically asks witnesses about psychiatric contact and obtains correct answers. Error from these sources would underestimate the association of violent death with mental illness.

Suicide is thought of as the main source of the increased mortality of the mentally ill. Our inquiry shows that death from causes similar to suicide but given undetermined and accidental verdicts contribute nearly as much again as suicide. Of the 412 deaths, 59% were suicides, 10% undetermined, and 31% accidental. Inquiry about the mortality of the mentally ill from unnatural causes should consider undetermined and accidental deaths as well as suicide.

How much does mental illness contribute to the total mortality of a population through its association with self-inflicted death? Deaths given one of the three verdicts collectively accounted for 1.6% of all deaths of Southampton residents aged over 15 for the eight years. Of this 1.6%, 56% had been treated by a psychiatrist and were presumably mentally ill at that time. Therefore mental illness is likely to have made a contribution of a little less than 1% to the mortality of the Southampton population. This estimate excludes from consideration as mentally ill the 50 deceased prescribed psychotropic drugs by family doctors shortly before they died and who had not been seen by a psychiatrist; including this 50 as mentally ill, increases the figure to 1.1%. This percentage is likely to be true for England and Wales because of the similarity of Southampton's population to that of the whole country.

The risk of death from unnatural causes, exceptionally high in the year following contact with a psychiatrist, was greatest for those aged 35–44 and least for those over 75. Black *et al* (1985) similarly reported finding the highest standard mortality rate (SMR) from suicide in the 30–49 age group and the lowest in those aged over 70. In-patients had a risk 2.3 times greater than that of out-patients.

An examination of the association between mental illness and violent and unnatural deaths with a potentially self-inflicted component for the population of a hospital catchment area for which a single psychiatric service is responsible has or is likely to become a part of clinical audit. Combining names acquired from the OPCS system with the local hospital's patient index to identify patients who have committed suicide or died from accidental causes is within the scope of the records department of a psychiatric service (King, 1983).

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Asperger's Syndrome: To Be or Not To Be?

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It has been questioned whether Asperger's syndrome (AS) is in fact a specific (high-functioning) subgroup of autism, rather than a distinct entity. Thirteen AS patients were compared with 13 autistic patients and 13 developmentally disordered controls. While there was symptom overlap between AS and autism, patients could be separated into one or other group. However, current criteria are based on symptoms, and it is argued that studies of genetics and treatment response are needed to elucidate the relationship between these developmental disorders.

In 1943, Leo Kanner described 11 patients with early infantile autism. A year later Hans Asperger (1944), an Austrian physician, described a group of children with a characteristic behavioural style that he described as autistic psychopathy. Asperger used the word 'psychopathy' to indicate that his patients' symptoms were indicative of character pathology. Patients of this type have since come to be diagnosed as having Asperger's syndrome (AS). While patients with infantile autism have been studied extensively, resulting in a rich literature on the subject, AS has received considerably less attention. Three bodies of literature exist on AS, one in German, a second in English, published primarily in Great Britain, and a third in Dutch. AS has received limited attention in the North American literature (Denkla, 1983; Paul & Cohen, 1984; Gillberg, 1985; Schopler, 1985; Volkmar *et al*, 1985; Nagy & Szatmari, 1986; Fisher *et al*, 1987).

van Krevelen (1971) discussed the differences between autism and AS, and a comprehensive review of this topic has been completed by Wing (1981). Recently it has been suggested that the distinction between AS patients and high functioning autistic patients is vague, and that empirical studies are necessary to establish the validity of AS as a separate condition (Schopler, 1985; Volkmar *et al*, 1985; Burd & Kerbeshian, 1987).

Is AS a useful diagnosis within the category of pervasive developmental disorders (PDDs)? Is it separable from the more established diagnosis of autism, or does it merely denote a specific subgroup of that entity? If AS is separable from autism, is it a distinct enough concept to demarcate it from the other PDDs? At this time sufficient data to address these questions are not available. This article reflects an initial effort to pursue these questions.

Present diagnostic criteria

Patients presenting with AS exhibit developmental abnormalities in several areas, as summarised by Wing (1981):

(a) pedantic lengthy speech
(b) stereotyped speech
(c) aprosodic speech
(d) impaired non-verbal communication
(e) peculiar social interaction
(f) lack of empathy with others
(g) repetitive activities
(h) resistance to change
(i) clumsy or stereotyped motor movements
(j) hypertrophied skills or circumscribed interests.

It appears that most of these patients have lucid speech well before four years of age, in contrast to the extreme speech delays and deviations noted in early infantile autism. Their speech is none the less abnormal. The prosodic elements are often markedly restricted. The content of their speech is lengthy and repetitive, often being focused on one area of interest. AS patients may exhibit intellectual abilities ranging from the moderately retarded to the gifted.

AS patients have generally very poorly synchronised non-verbal communication. Their gestures lack the rate, rhythm, and integration of the normal movements which supplement spoken language. These patients often use very broad, intrusive gestures, or not use subtle non-verbal communication.

Patients with AS seem to desire interpersonal involvement but are socially inept, having limited ability to monitor others' reactions to their social ventures. They often attempt to channel all conversation into their areas of interest. They frequently have difficulty with respect to others' personal space, and approach too closely without being aware of any resulting discomfort.

Most patients with AS have areas of very intense preoccupation. These may involve topics such as electronics, outer space, animal species (e.g. dinosaurs), or increasingly, an extreme fascination with computers. They may often develop considerable skills in these areas: an AS patient may for example be able to take apart and reassemble radios or to use computers very readily.

In this article, a case history is presented. Thirteen AS patients are compared with 13 with autism and 13 developmentally disordered controls. The utility of separating AS from other PDDs is discussed, and a tentative rationale for using AS as a descriptive modifier of the subtypes of PDD is proposed as a schema needing further consideration. Some of these AS patients have been described previously in other articles (Kerbeshian & Burd, 1986; Burd *et al*, 1987*a*; Fisher *et al*, 1987; Burd & Kerbeshian, 1987).

Case presentation

XH was born after a normal pregnancy, labour and delivery. Early on, his mother did note some problems with relatedness; however, XH was not a stiff or avoidant child. He engaged in body rocking and was a head banger. He walked at 11 months and was toilet trained at two and a half. After three years of age he was quite content to play by himself for long periods. There were episodic problems with overactivity. He experienced a rather abrupt onset of speech, not talking at all, and then suddenly talking in phrases. Neither echolalia nor pronomial reversals were ever noted. He had very close relationships with his mother and an older sister in spite of his peculiar interpersonal style and his deficient capacity for empathy. By four years of age he was noted to be odd compared with his peers.

Over the years he developed a number of stereotypic mannerisms and a compulsive preoccupation with various themes and ideas (the day's events, and computer technology, particularly since he learned that cartoon figures could be produced on a computer screen). He has a dishevelled appearance and makes intrusive contact with others. He exhibits a number of motor and vocal tics as well as complex, compulsive mannerisms. These are suppressible and have waxed and waned over several years. He is studying music at university, and is doing reasonably well academically.

XH continues with what he refers to as his "G spots". These are areas of active involvement with which he becomes obsessed. He becomes so involved in these areas that even minor frustrations cause him anguish. He is noted to have aprosodic speech, with periodic grunts and repetitive deep respirations. His non-verbal communication is asynchronous and gesturing is exaggerated. He has few friends but will occasionally go out with other students. He is aware that he is unable to maintain friendships, and that it is difficult for him to sustain conversation and interaction with others. He states that he feels the most comfortable when he is by himself, using his computer. His WAIS full-scale IQ is 129. His diagnoses are atypical pervasive developmental disorder with Asperger symptoms and Tourette's disorder.

Method

Wing's (1981) criteria (listed above) for AS are broadly descriptive and do not specify thresholds to make the diagnosis. A further step towards establishing at least the content validity of AS would be a comparison with a similar clinical entity with a degree of overlap. Because theorists have argued that AS is not clinically distinct from autism, we chose a cohort of 13 autistic patients as a comparison group and a cohort of 13 general clinic referrals (without PDDs) as controls.

Subject selection

All the comparison and control patients in this study were first diagnosed within the framework of DSM-III by a child psychiatrist. These diagnoses were then reviewed and corroborated by a multidisciplinary team, including a psychologist. As AS does not appear in DSM-III (American Psychiatric Association, 1980), this was diagnosed in a way similar to that described by Volkmar *et al* (1988), adopting Wing's (1981) descriptive conception of the condition.

Over four years more than 1300 patients with mental and/or developmental disorders were seen in the authors' direct care and consulting practices, and were scrutinised for a clinician's diagnosis of AS. Every patient with a provisional diagnosis of a PDD and a substantial proportion of the patients with other developmental problems were seen by a child psychiatrist who formulated a diagnosis using DSM-III criteria.

The autistic patients were selected from the state evaluation centre's registry of 37 patients with DSM-III-defined infantile autism. The highest functioning individuals who could be matched by age and sex with AS patients were identified. We also attempted to match the AS and autistic patients by IQ, but very few autistic patients scored above the borderline level (IQ of 70–80).

The control group patients were selected from the centre's population of developmentally disordered children who did not have a diagnosis of either autism or AS. These patients were seen at the state's comprehensive evaluation centre. These patients had specific learning disabilities, hyperactivity, behaviour disorders, and mental retardation. These patients were also matched with the AS patients for age, sex, and IQ.

TABLE I
Means, standard deviations and F *statistics for age and IQ of patients with Asperger's syndrome, autism, and controls*

	Asperger's syndrome	*Autism*	*Controls*	F *value*	*Significance (P)*
No. of males/females	11/2	11/2	11/2	–	–
Mean age (s.d.): years	16.5(6.2)	13.2(6.8)	15.8(5.6)	1.04	0.36
Mean IQ (s.d.): WAIS	86.7(19.3)	44.3(21.4)	82.2(23.3)	15.38	<0.001

Results

Table I presents *F* statistics for comparison of age and IQ among the patients with AS, autism, and controls.

Each of the 13 patients conforming to Wing's (1981) concept of AS also had a DSM-III-defined PDD. While two of 13 AS patients had met criteria for DSM-III-defined autism at one time, 85% had never met these criteria (Table II). These 11 patients were felt to exhibit developmental deviation sufficient for a diagnosis of atypical PDD.

Of all patients seen over the four years, AS was diagnosed in 17% of non-autistic patients with a DSM-III-defined PDD and 5% of DSM-III-defined autistic patients. In the authors' experience, DSM-III-defined autistic patients who develop AS appear to develop higher IQs, more functional communication, and improved interpersonal skills when contrasted with autistic patients who did not later conform to Wing's concept of AS as interpreted by a clinician's diagnosis.

TABLE II

DSM-III criteria for autism applied to 13 Asperger's patients: (+) patients' symptoms met DSM-III criteria, (−) patients' symptoms did not

Sex	F	M	M	M	M	M	F	M	M	M	M	M	M
Onset before 30 months of age	+	+	+	+	−	−	+	−	−	−	+	+	−
Pervasive lack of responsiveness	+	+	−	−	−	−	−	−	−	−	−	−	−
Gross deficits in language development	+	+	−	+	−	−	+	−	−	−	−	+	−
Peculiar speech if present	+	+	+	+	−	−	+	−	−	−	+	+	+
Bizarre responses to environment	+	+	+	−	−	−	+	−	−	−	−	+	−
Absence of delusions and hallucinations	+	+	+	+	+	+	+	+	+	+	+	+	+

TABLE III

The number and percentage of patients with Asperger's syndrome (n = 13), autism (n = 13) and controls (n = 13) who at any time met the various criteria for each condition

	Asperger's syndrome		*Autism*		*Controls*	
	Frequency	*%*	*Frequency*	*%*	*Frequency*	*%*
Criteria for AS						
Pedantic, lengthy speech	8	62	0	0[1]	0	0
Stereotyped speech	11	85	9	69[2]	4	31
Aprosodic speech	10	77	5	38[2]	2	15
Impaired non-verbal communication	13	100	7	54[3]	2	15
Peculiar social interaction	13	100	13	100	2	15
Lack of empathy for others	12	92	13	100	0	0
Repetitive activities	11	85	13	100	5	38
Resistance to change	9	69	13	100	0	0
Clumsy or stereotyped motor movements	13	100	13	100	4	31
Hypertrophied skills or circumscribed interests	12	92	4	31	1	8
Criteria for autism						
Onset before 30 months of age	13	100	13	100	-	-
Pervasive lack of responsiveness	2	15	13	100	-	-
Gross deficits in language development	5	38	13	100	-	-
Peculiar speech if present	8	62	10	77[2]	-	-
Bizarre responses to environment	5	38	13	100	-	-
Absence of delusions and hallucinations	13	100	13	100	-	-

1. None of the autistic persons were verbally fluent enough to permit comparison.
2. Three of our autistic patients lacked or had significant poverty of speech.
3. Non-verbal communication used in a restricted sense. Six autistic patients had pragmatic speech impairment to the point that non-verbal communication could not be judged meaningfully.

Intergroup comparisons

The six DSM-III criteria for autism were applied to the 13 AS patients and the 13 autistic patients. Wing's ten criteria for AS were then applied to the 13 autistic patients, the 13 controls, and the 13 AS patients (Table III). The application of Wing's criteria to the autistic group was difficult at times owing to the developmental specificity of some of those criteria. As a result, some of the intergroup differences appear curious at first glance. For example, one finding indicates that only 54% of the autistic group have impaired non-verbal communication. In this study, non-verbal communication was interpreted as 'metacommunication', that is, those aspects of vocal intonation and gesture which supplement speech. This is similar to the definition provided by Wing (1981), and later used in a study of AS by Scott (1985). By this definition, an autistic person would first have to have some meaningful speech in order to exhibit impaired non-verbal communication.

As can be seen in Table III, pervasive lack of responsiveness is much more common among autistic patients, as are bizarre responses to the environment and gross deficits in language development. The symptoms most specific to AS are pedantic, lengthy speech, hypertrophied skills or interests, and aprosodic speech. While there is symptom overlap between AS and autism, it appears that patients can be separated into one or the other condition. Major symptom discriminators for a diagnosis of autism were the presence of pervasive lack of responsiveness and the absence of pedantic, lengthy speech; and for AS the presence of hypertrophied skills or circumscribed interests and the absence of pervasive lack of responsiveness.

The patients with AS were even more distinguishable from the control group. As can be seen from Table III, 60% or more of the AS patients met each of the criteria, whereas very few of the control patients met any of these criteria. The presence of specific AS symptoms among the control group appeared to be randomly distributed.

Discussion

This study attempted to determine whether DSM-III-defined autism and AS are different syndromes or whether they represent a behavioural continuum of a single disorder (autism). Every attempt was made to identify high-functioning patients who met DSM-III criteria for infantile autism. The group identified for matching by sex and age constituted the 13 highest-functioning autistic patients from the 37 patients meeting DSM-III criteria for infantile autism. In this population a satisfactory match in IQ between our autistic and AS patients could not be achieved (Table I). The DSM-III criteria for infantile autism are quite specific, and it appears that higher-functioning patients rarely meet these criteria. The overlap between the autistic group and the AS group is notable, especially when compared with the control group. In order to ascertain whether AS is a separate nosological entity from autism or is truly a 'good-prognosis autism', the variable of IQ should be properly controlled for. In this study we were unable to accomplish this, perhaps because of the very nature of DSM-III criteria for autism. It is important to realise that these are patients in a geographically defined population which has been carefully searched for all children meeting criteria for DSM-III-defined PDDs.

Although Asperger symptoms may not crystallise fully in many patients until roughly four years of age, all our patients exhibited developmental difficulties before 30 months. As a result, they did not meet the criteria for childhood-onset PDD in DSM-III. The classification of AS as part of the spectrum of atypical PDD or in two cases as residual autism seems most appropriate, as it precludes a premature judgement as to whether the syndrome is truly valid in its own right. To attempt to separate our group of AS patients from the spectrum of atypical PDD using a similar method of symptom comparison would have been most difficult, as atypical PDD is a residual category, without specific symptom demarcation. Although our two patients who at one time met full criteria for autism may represent a subgroup of AS patients whom one may define as 'good-prognosis autistics', the majority (11 of 13) at no time met DSM-III criteria for infantile autism. Because of the changes in the criteria for PDDs in DSM-III-R (American Psychiatric Association, 1987), it is difficult to determine what the results of the current study would have been if these criteria had been applied.

Differences found between autistics and AS patients may be due to the defining characteristics, rather than inherent differences in the groups. For example, those with DSM-III-defined autism demonstrate gross defects in language development. They therefore would be incapable of showing the pedantic and lengthy speech of AS. The differences apparent between the two groups in terms of this factor might very well be tautological. A meaningful comparison based on symptom criteria would be greatly enhanced if each group could also be defined by non-symptom externally validating criteria.

It is noteworthy that clinicians who have argued against the inclusion of AS in classification systems, such as DSM-III-R, have stated that patients identified as having AS are actually high-functioning autistic patients (Schopler, 1985; Volkmar *et al*, 1985). If such is the case, behavioural items in AS may be basically the same as those in autism, but at a more advanced, though none the less deviant, level of development. For example, circumscribed interest patterns may be a higher-level manifestation of resistance to change. The observation that the majority of these patients never met criteria for autism applies only to DSM-III-defined autism. Such might not be the case for autism defined by some externally validating characteristic such as familial transmission, a biological marker, or clinical course.

It is the authors' opinion that the problems presented in these AS patients do not reflect a personality disorder, as they appear to represent a distinct evolution of developmental difficulties which are first manifested early in life. The coexistence of Tourette's syndrome in some of these AS patients has been elaborated upon elsewhere (Kerbeshian & Burd, 1986), as has the coexistence of Tourette's syndrome and PDD (Burd *et al*, 1987*b*).

One could argue that these patients also meet criteria for either schizophrenia, schizotypal personality, or schizoid personality (Wolff & Barlow, 1979; Wolff & Chick, 1980). The absence of hallucinations or delusions, and the early onset of pervasive difficulties across multiple developmental lines in a pattern not mediated primarily through psychodynamic conflict, make these less plausible nosological possibilities.

Table III has outlined the many similarities and the notable differences between the autistic and AS patients. Although of some interest in demarcating between the two groups, these differences suggest no clear conclusions. It is important to continue to study the syndromal validity of AS. The issue of the sensitivity of DSM-III criteria for diagnosing autism has already been addressed (Volkmar *et al*, 1988). Although these criteria appear to be quite specific for autism, they may be more sensitive to 'bad-prognosis autism', and less sensitive to 'good-prognosis autism', perhaps as represented by AS.

More fruitful results may accrue from longitudinal studies of children through adolescence and adulthood using published criteria such as those from Wing (1981). Genetic and family studies using shared biological markers in children, adolescents, and adults who meet criteria for various PDDs would also be very helpful in clarifying the validity of AS. In future studies we hope to ascertain whether differences between autism and AS can be demonstrated in aetiology, response to treatment, and outcome. In these studies both DSM-III and DSM-III-R criteria will be used to facilitate the comparability of past and future results.

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Mentally Retarded Criminal Offenders in Denmark

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Based on data from the Danish Central Criminal Register, the total number of mentally retarded offenders serving statutory care orders on a census day decreased from 290 in 1973 to 91 in 1984. The reduction was caused by shorter sentences and a dramatic decrease in the number of sentenced borderline retarded offenders; the total number of sentences per year slightly decreased, and the number of first-time sentences was stable. Crimes of property are decreasing among this population, while violence, arson, and sexual offences are increasing. Behaviour disorder was found in 87.5% of 91 offenders serving care orders in 1984. Offensive behaviour was significantly predicted by early institutionalisation, having retarded or divorced parents of low socio-economic status, and behaviour disorder of social–aggressive type. Independent significant effects were attributed to behaviour disorder and low socioeconomic background. All predictors were closely correlated. Biological factors did not have any significant predictive value.

The relationship between mental retardation and criminality has been of great interest to criminologists since the beginning of the century. From the early, rather oversimplified opinion that "the greatest single cause of delinquency and crime is low grade mentality, much of it within the limits of feeble-mindedness" (Goring, 1913; see also Goddard, 1920; Sutherland, 1931), our understanding of the topic has gradually improved (Woodward, 1955; Hurwitz & Christiansen, 1968; Walker & McCabe, 1973; Bluglass, 1980). Our current understanding of this relationship can be summarised as follows:

- (a) there is a slightly increased prevalence of the mildly and borderline retarded among offenders
- (b) there is a slightly increased incidence of crime among the mildly and borderline retarded
- (c) the higher prevalence should be expected because of the higher risk of detection for this population
- (d) there is a clearly decreased incidence of crime among the severely and profoundly retarded
- (e) in relation to other offenders there are more arsonists and sexual offenders among mentally retarded criminals
- (f) borderline retarded offenders exhibit more recidivism.

The services for the mentally retarded have undergone great changes, dominated by normalisation and deinstitutionalisation. It has been reported that the number of care orders is rapidly decreasing; the possible influence of these changes in service provision on the occurrence of crime, is, however, unknown. This study describes trends in service provision for mentally retarded offenders over 1970–84, and analyses social and psychiatric factors as predictors of crime in the mentally retarded.

Method

Offenders who are held irresponsible because of mental retardation at the time of the crime are, in accordance with the Danish Penal Code, not punished. Paragraph 68 of the code allows for either a care order without compulsory admission (community care order) or detention in an institution. Both types of care order are indeterminate in length and are terminated only after by a subsequent court ruling on review of a particular case. Care orders with compulsory detention in an institution are usually changed to a community care order, rather than being terminated directly. For the period 1 January 1970–31 December 1983, the Danish Central Criminal Register was inspected, and information found on 274 mentally retarded persons receiving care orders. The register gives demographic data (age, sex, date and place of birth) for all offenders.

Further information on these 274 offenders was obtained from the files of the Danish Medico-legal Council. These files contain statements on the mental health of the offenders based on a statutory psychiatric examination.

The sample of 274 persons was subdivided for the purposes of analysis:

- (a) 91 offenders with a valid sentence on a census day (1 January 1984) were compared with 290 offenders from an almost identical Danish survey study from 1973 (Betænkning, 1982; census day 15 January 1973)
- (b) 66 offenders first sentenced between 1 January 1970 and 31 December 1973 were compared with 57 offenders first sentenced in a similar period ten years later (1 January 1980–31 December 1983)
- (c) 83 offenders sentenced between 1 January 1970 and 31 December 1974 were followed up for ten years, using the register, in order to analyse recidivism

(d) 72 of the 91 offenders from group (a) accepted a follow-up personal interview; these 72 offenders were compared with 72 matched controls in order to find predictors of criminal behaviour.

From the register search 88 persons under a current care order on 1 January 1984 were found. On application to the local authorities responsible for treatment, this total was confirmed. However, the latter inquiries revealed a further three offenders meeting the study criteria. These three were originally found on the register but had been misclassified as released. The fact that information on 88 of the 91 was correct indicates a high internal validity of the register. One problem however is that crimes such as violence against staff members or other patients are rarely reported.

The 19 offenders who refused to take part in the follow-up study (d) did not differ significantly ($P<0.05$) from the rest with respect to age, sex, or degree of retardation. The remaining 72 (79.1%) were all interviewed in their homes or institutions by the author between 1 January 1987 and 30 April 1988. The Medical Research Council's Schedule of Handicaps, Behaviour and Skills (MRC–HBS) and a psychiatric check-list (Lund, 1985) were used.

From a community-based sample of 302 mentally retarded adults elaborated by epidemiological criteria for use in a prevalence study of psychiatric morbidity, 72 controls were chosen, matched in pairs by sex, age, and degree of retardation. These 72 non-criminal controls had been interviewed earlier by the author, also with the MRC–HBS schedule and the same psychiatric check-list.

Information on social and biological factors was collected from the case records and legal documents.

Statistical analyses

Odds ratio (OR) and corresponding χ^2 values were calculated using bivariate techniques for predictors. Predictors significant at the 95% level were processed further using multivariate logistic-regression analysis. The estimated relative risk for a given outcome is the product of the adjusted or independent OR of the individual predictors (Jørgensen & Aagaard, 1988). The goodness of fit of the model is expressed by the likelihood ratio, G^2. The amount of information added by the individual predictors is estimated by the increase in G^2. The amount of information added by the individual predictors is estimated by the increase in G^2, ΔG^2 (1 d.f.) after omitting the predictors one at a time. The information per cent (I) of the individual predictors is the square of the correlation coefficient, calculated as the sum of the squares of the normalised means divided by the number of patients minus one. A level of significance of I can be calculated, and all I values reported have $P<0.05$. The maximal information per cent (I_{max}) is the square of the multivariate correlation coefficient. After the implementation of the logistic analyses the predictors still recoded as 1/0 were further processed through a matrix of intercorrelations. Further information on the statistical procedures is given by Ipsen & Feigel (1966), Bishop *et al* (1975) and Fleiss *et al* (1986); for earlier examples of application of the procedures see Jørgensen & Aagaard (1988).

Results

Of the 274 offenders only 17 (6.2%) were women. The offences committed by women were generally less violent and community care orders were used more frequently than institutional detention. Because of the small number of women, no further sex ratios are presented.

The total number of care orders per year decreased slightly over the study period, from an average of 40 per year to 29. However, first-time orders remained stable, at approximately 14 per year, or 3.1 per million population aged over 15, per year.

(a) Comparison of 1973 and 1984 data

The decline from 290 offenders in 1973 to 91 in 1984 (Table I) was caused by the dramatic decline in numbers

TABLE I

Mentally retarded criminal offenders serving community or institutional care orders on a census day in 1973 and 1984: comparison of degree of retardation and duration of term

	1973	*1984*				
	(n = 290)	*All (n = 91)*	*Institution (n = 41)*	*Community care (n = 50)*	*OR (95% c.i.)*[1]	
Degree of retardation						
Borderline: *n*(%)	155 (53.3)	3 (3.3)	2 (4.9)	1 (2.0)	0.03	(0.01–0.06)
Mild: *n* (%)	109 (37.6)	78 (85.7)	34 (82.9)	44 (88.0)	10.1	(5.8–17.7)
Moderate and severe: *n* (%)	26 (9.0)	10 (11.1)	5 (12.2)	5 (10.0)	1.3	(0.5–3.2)
Duration of term						
0–23 months: %	21.4	48.4	61.0	38.0	—	
24–59 months: %	24.8	40.7	34.1	46.0[2]	3.4	(2.1–5.5)
60+ months: %	53.8	9.9	2.4	16.0	10.6	(5.7–19.9)

1. Odds ratios for 1984 v. 1973.
2. Community versus institutional care 1984, ≥2 years, OR = 2.5 (1.1–5.7).

TABLE II

Mentally retarded offenders receiving first sentences in 1970–83 by degree of retardation and type of crime (%)

	1970–73 (n=66)	*1980–83 (n=57)*	*Odds ratio: 1970–73 v. 1980–83 (95% c.i.)*
Degree of retardation			
Borderline	33	6	0.1 (0.04–0.3)
Mild	31	45	4.5 (2.1–9.8)
Moderate and severe	2	6	3.9 (0.8–18.3)
Type of crime			
Property	68.2	29.2	0.3 (0.1–0.6)
Violence	7.6	24.6	2.3 (0.8–6.2)
Arson	10.6	36.8	5.1 (1.8–14.7)
Sex	15.2	31.6	1.8 (0.8–4.3)

in the borderline group. Another contributing factor was the significant reduction in the length of care order served (Table I). At the 1984 census only one person had stayed in hospital for over five years, while this was frequently found in 1973. In 1984 care orders served in the community were longer on average than those served in institutions.

The type of crime had also changed. Offenders sentenced in 1984 had more frequently committed crimes of violence and arson while crimes of property were reduced. Unfortunately it was not possible to carry out stratified analysis for degree of retardation, because the relevant data for 1973 were no longer available.

(b) Offenders first sentenced 1970–73 and 1980–83

The number of borderline retarded persons receiving their first sentence in 1980–83 was significantly lower than those first sentenced in 1970–73, but this was almost counterbalanced by a more than 50% increase in the number of more retarded offenders. The change in the type of crimes leading to first sentences could be stratified for degree of retardation, and the results are shown in Table II.

(c) Ten-year register follow-up

Recidivism was found in 60 of the 83 offenders followed up on the Central Criminal Register ten years from their first sentence in 1970–74. The criminal careers were very different: 35 had received only one sentence (the index) but of these, 12 were registered with the police for further criminal activity not serious enough for the police to pursue prosecution; 19 received care orders with compulsory detention for repeating serious offences such as arson and sexual crimes; 29 offenders, mainly of borderline retardation, repeated crimes of property. For the latter group, service provision was problematic because of their antisocial behaviour and relapse into criminal activity, and eventually the court passed gaol sentences.

TABLE III

Mentally retarded criminal offenders serving community or institutional care orders on 1 January 1984, by age: comparison with all retarded individuals on the Danish Retardation Register

Age: years	*Register (n=9547)*	*Offenders (n=91)*	*OR (95% c.i.)*
15–19	20.0	2.2	0.1 (0.03–0.3)
20–29	30.2	26.4	0.8 (0.5–1.4)
30–39	17.7	40.7	3.2 (2.1–4.8)
40–49	12.8	15.4	1.2 (0.7–1.9)
50–59	11.5	14.3	1.3 (0.7–2.4)
60+	9.8	1.1	0.1 (0.02–0.5)

Relapse into the same type of crime as committed at the time of the first sentence was frequent only for crimes of property; of 29 offenders committing either arson, violence or sexual offences at first sentence only seven repeated such crimes. However, transition to other forms was frequent.

There was a clear, but not significant, tendency for repetitive sentences to community or institutional care, and for changes to imprisonment, to occur most frequently in: borderline patients (OR 2.4 (1.05–5.9)); offenders with criminal careers before first care order (OR 2.2 (0.8–6.0)); and offenders aged 20–29 years at the time of first sentence (OR 1.6 (0.8–3.8)).

(d) The 1984 census and follow-up

Only six of the 91 offenders with a valid sentence on 1 January 1984 were women. Again there was a tendency to non-violent crimes and less institutionalisation in the small group of women.

The median age was 34 years (range 17–74 years), s.d. 10.8 (Table III). The number of very young and old offenders was low compared with all retarded, while a preponderance of offenders were aged 30–39 years (Table III). Mild retardation was the dominating level of handicap in the offender group at a prevalence of 85.7% (Table II), compared with 27.5% in the general population of the mentally retarded. The majority (68.1%) were living in institutions at the time of the investigation, but 25.3% were living alone or with a partner. The offenders were well known to the services before committing their first crime; in fact only 10 (11.0%) had not been in care before. At the time of the present offence, 67 (73.6%) were under a care order, and 29 (31.9%) were living in an institution.

The crimes committed by the mentally retarded are very variable in type, reflecting compulsion or expression of mood (Table IV). Only a third were registered for only one type of crime. Mentally retarded offenders usually act alone, although occasionally they may follow along in a group committing crimes of property. Only seven offenders were influenced by alcohol at the time of crime, and none appeared to be under the influence of drugs. There were no cases of attempted suicide in connection with the crime.

TABLE IV
Types of crime committed by mentally retarded criminal offenders serving care orders on 1 January 1984

	Sentence on census day (n = 91)				*All sentences* (n = 91)			
	Total		*Alone*[1]		*Total*		*Alone*[1]	
	n	%	n	%	n	%	n	%
Property	38	41.8	18	19.8	58	63.7	8	8.8
Violence	21	23.1	10	11.0	34	37.4	4	4.4
Arson	32	35.2	24	26.4	45	49.5	13	14.3
Sex	19	20.9	11	12.1	31	34.1	7	7.7
Other penal code	4	4.4	—		5	5.5	—	
Traffic offences, etc.	9	9.9	2	2.2	11	12.1	1	1.1
Total			65	71.5			33	36.3

1. Offenders committing one type of crime only.

Interview study

Of the 91 offenders with a valid sentence on 1 January 1984, 72 agreed to participate in a personal interview. The other 19 did not differ significantly (95% level) with regard to demographic and forensic data.

Behaviour disorder was found in 63 (87.5%), while only few other psychiatric disorders were present (Table V). Compared with 302 randomly selected mentally retarded adults (Lund, 1985), the prevalence of behaviour disorders is much higher in the offender group (even when stratified for sex, age, and degree of retardation). The behaviour disorders were mainly of social–aggressive type and most often present since childhood or early adolescence. Their constant hyperactive, rebellious and meddling attitude had dominated their personal relationships, leading to defeat and rejection. Criminal recidivism was frequent. According to information from staff members and case records, 48 of the 72 had committed crimes during the year before the interview (including 6 violent, 16 arson, 5 sexual offences). Less than half of these offences were reported to the police. The frequency of recidivism was significantly increased in the persons with a behaviour disorder (OR 5.0 (1.2–20.1)). Recidivism of violent and sexual offences was found only in the behaviour disorder group.

TABLE V
Prevalence of psychiatric disorders in mentally retarded offenders, compared with randomly selected non-criminal retarded adults

	Randomly selected mentally retarded adults (n = 302)		*Mentally retarded offenders* (n = 72)	
	%	*(95% c.i.)*	*%*	*(95% c.i.)*
Schizophrenia	1.3	(0.0–2.6)	1.4	(0.0–4.1)
Affective disorders	1.7	(0.2–3.2)	2.8	(0.0–6.6)
Dementia	3.6	(1.5–5.7)	—	
Infantile autism	7.6	(4.6–10.6)	—	
Other psychoses	1.0	(0.0–2.1)	—	
Neuroses	2.0	(0.4–3.6)	—	
Behaviour disorder	10.9	(7.4–14.4)	87.5	(79.9–95.1)
Totals	27.1	(22.1–32.1)	91.7	(85.3–98.0)

Predictors

Bivariate distribution of the data showed all social items and behaviour disorder to be significant predictors of crime (Table VI). No significant differences were found in biological factors, such as epilepsy (OR offenders epilepsy ≤10 years 1.5 (0.6–3.5)) or cause of mental retardation, except that pre-natal or post-natal brain damage was more frequent in the offender group (OR 2.2 (0.8–6.1)) while chromosome abnormalities were most frequent in the control group (OR 2.7 (0.9–8.5)). Seven patients with Down's syndrome were found in the control group (OR 7.6 (1.2–47.0)) compared with only one in the offender group (a severely retarded young man with a fascination for arson who eventually set fire to the family's flat and inadvertently burnt his father to death). In the offender group there were three persons with a sex chromosome abnormality compared with one in the control group.

Psychiatric disorder, except for behaviour disorder, was found in three patients in the offender group (one schizophrenic, two with affective disorder) and in six in the control group (one schizophrenic, one affective disorder, two infantile autism, two with neurosis).

In both the bivariate and the logistic model (Table VII) behaviour disorder is the all-important predictor. Of the social items, only low socioeconomic background ($P < 0.05$) and retarded parents ($P < 0.10$) are assigned independent predictive value. The bivariate significant predictors are closely correlated (all with $P < 0.05$).

When behaviour disorder is chosen as the dependent variable (Table VI), four of the five social items are significantly associated with it. Again, no biological factors turned out to be significant predictors. In the logistic model (Table VII) only early institutionalisation has significant predictive value ($P < 0.01$). Again, the predictors were closely intercorrelated (all with $P < 0.01$). The informative value, expressed by I_{max}, was low for the specific predictors of behaviour disorder.

From the model it is possible to calculate the individual probabilities of outcome by use of adjusted and raw OR

TABLE VI

Mentally retarded offenders compared with non-offenders matched by sex, age, and degree of retardation: social and psychiatric factors as predictors of crime and of behaviour disorder

	Offenders[1] *(n = 72)*	*Non-offenders*[1] *(n = 72)*	*OR (95% c.i.)*	*Behaviourally disordered (n = 68)*	*Not behaviourally disordered (n = 76)*	*OR (95% c.i.)*
One or both parents retarded[2]	14	4	4.1 (1.4–12.3)	11	7	1.9 (0.7–5.1)
Fathers' socioeconomic status low[3]	57	33	4.5 (2.2–9.2)	51	39	2.8 (1.4–5.6)
Parents separated before the child was 5 years old	25	6	5.9 (2.4–14.6)	24	7	5.4 (2.3–12.9)
Institutionalised ≤ 5 years of age	27	5	8.0 (3.2–20.7)	26	6	7.2 (3.0–17.4)
No. of institutions ≥ 3 before the age of 7	19	3	8.2 (2.8–24.4)	19	3	9.4 (3.1–28.2)
Behaviour disorder	63	5	93.8 (35.5–228.7)			

1. Matched in pairs by sex, age, and degree of retardation.
2. As indicated by a search through the Danish Retardation Register.
3. Social group 5 (of five).

TABLE VII

Logistic regression analysis for the most informative predictors of crime and behaviour disorder in the mentally retarded

	OR	ΔG^2	I
Predictors of crime			
Behaviour disorder	119.4	89.29	65.09
Low socioeconomic background	4.6	4.94	11.85
Parents retarded	5.5	3.63	4.40
Institutional care before 5 years of age	1.9	0.49	13.50
Parents separated	1.2	0.04	10.30
Weighted OR	0.03		
G^2	9.67	15 d.f.	$P>0.80$
I_{max}	68.37		
Predictors of behaviour disorder			
Institutional care before 5 years of age	4.14	7.30	13.27
Parents separated	2.34	2.47	10.04
Fathers socioeconomic status low	1.70	1.33	5.97
Weighted OR	0.41		
G^2	8.56	4 d.f.	$P>0.05$
I_{max}	16.58		

values. Thus, if a person has a father who was retarded, if he was institutionalised before the age of five, and if he eventually developed a behaviour disorder, his risk of receiving a care order for an offence is 97%:

$$\frac{0.03\times5.5\times1.9\times119.4}{1+(0.03\times5.5\times1.9\times119.4)}=0.97$$

Discussion

The reduction in number and change in type of care orders placed upon mentally retarded offenders at sentencing in Denmark, reflects a general trend towards normalisation and deinstitutionalisation for the mentally handicapped population as a whole. This has not been reported previously, although an impression of similar developments has been reported (e.g. McLaughlin, 1988), especially in Scandinavia.

There is no reason to believe that the immense reduction in the number of borderline retarded offenders receiving care orders reflects a genuine reduction in criminal activity in this population. Rather, it is suspected that ordinary penal sanctions are used in their stead. The follow-up of the present study seems to confirm this. Part of the background for this shift could be the fact that institutions and other care facilities for the retarded are now mainly organised for and populated by more severely retarded patients. The borderline patient no longer fits into these settings.

The shorter terms served under care orders are a major improvement. Often patients were kept in custodial care for five to ten years or more after trivial crimes. The prediction of dangerousness is very difficult, but seems to be handled with great skill: the shorter terms have not resulted in increasing recidivism of severe crimes.

The increasing number of mildly retarded offenders receiving first sentences is not alarming, as the numbers are still small. However, this could be a result of deinstitutionalisation and therefore calls for careful handling of potential offenders. Unfortunately, very little is known about predictive factors for potential offending in this population.

There are however predictive factors for those with an established pattern of offending. A young mildly or borderline retarded person with low socioeconomic background, dominated by inappropriate parental performance and, frequently, early institutionalisation, is at great risk for the early development of an aggressive behaviour disorder, leading to delinquency. Similar offender characteristics were found by Day (1988) in his study of 20 males in a hospital-based treatment programme for handicapped offenders. No significant differences in prevalence of somatic complications was observed, although mental retardation caused by pre-natal or post-natal brain damage was frequent in the offender group, while autosomal chromosome aberration in the form of Down's syndrome was significantly more prevalent in the control group. The effect of confounders is minimised by the use of matching in pairs and stratified analysis in the form of multivariate logistic regression.

These risk factors are not surprising, in fact they have been demonstrated in many earlier investigations of non-retarded samples (Mulligan *et al*, 1963; Jonsson, 1967; Robins, 1970; Aagaard, 1986). It can therefore be concluded that the causes of delinquency in the mentally retarded seem to be the same as in the non-retarded.

These results could give guidance in prevention and treatment. The majority of retarded offenders are doubly disabled by the combination of behaviour disorder and intellectual handicap. As normal penal sanctions have not proved at all effective, long-term treatment plans should be elaborated to tackle persistent behaviour disorders. In most cases the risk of criminal recidivism cannot be expected to decrease within a given time, and the offenders should therefore be followed for some years.

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A Study of Psychotropic Medication Given 'As Required' in a Regional Secure Unit

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Within the setting of a regional secure unit, all doses of medication given *p.r.n.* over three months were ascertained and the details of each administration determined from prescription charts and a semistructured interview with the nursing staff involved. Thirty-two patients were resident for all or part of the study, all compulsorily detained. Only 15 were given medication *p.r.n.*; compared with the other patients they were significantly younger and more likely to be detained under civil orders than under the criminal provisions of the Mental Health Act 1983. Rarely was the medication the sole intervention in a crisis. In terms of effects on the overall treatment programme of the patient, medication given *p.r.n.* seemed to have an impact in only one small subgroup.

Psychotropic medication is widely prescribed and administered on an 'as-required' (*p.r.n.*) basis. Policies on its use, however, vary considerably between different units: one hospital in Northern Ireland gave medication *p.r.n.* after only six of 208 incidents among disturbed women over six months in 1981 (Cooper *et al*, 1983). There is a growing literature on the characteristics of violent psychiatric patients (Taylor, 1986) and some of this has dealt more specifically with the psychiatrist's pharmacological response to such violence (Tardiff, 1981; Appelbaum *et al*, 1983). However, little has been written about the circumstances surrounding the administration of emergency medication, the features of the patients who receive it, and its relationship to other management strategies. In this study a preliminary attempt is made to describe the use of medication given *p.r.n.* within the setting of a regional secure unit.

The Denis Hill Unit is a 30-bed facility for men and women which acts as the central clinic in the South East Thames Region forensic psychiatry service. It accepts patients for rehabilitation from the special hospitals, but more than three-quarters of the patients are admitted from prison or general-hospital units, generally in an acutely disturbed state. At any one time approximately two-thirds of the patients on the unit exhibit psychotic symptoms. On the interim secure unit, which was its predecessor, violence was the most powerful patient variable determining admission (Offen & Taylor, 1985), and the only change in admission patterns since then has been a decrease in the proportion of special-hospital rehabilitation cases.

Method

At the start of the study there were 17 patients on the unit, rising to 26 three months later. Thirty-two patients were resident for all or part of the study period (103 days). All were included in the study for some comparisons, but most data were from the 15 who had received one or more doses of psychotropic medication given *p.r.n.*

Each patient on the unit had his or her own prescription card, with a special section for medication given *p.r.n.*, in which every dose was recorded. One of the two principal researchers (FWAB, SMcL) identified all patients receiving psychotropic medication *p.r.n.* (other than hypnotics) during the three months, by examining all the prescription cards used. In addition, for 73 randomly selected days of the study period, administration of such medication was examined in detail. One of the two same researchers checked each card on these days and interviewed any member of the nursing staff who had decided to administer medication *p.r.n.* in the previous 24 hours. Their responses were recorded on a standard check-list devised for the study. Particular attention was paid to nursing interventions attempted before and after the medication, as well as the patient's mental state immediately before and 30 minutes after its use. In relation to behaviour, signs (see Table IV) were recorded as being present or absent, in the judgement of the nurse attending the patient.

The data were analysed using the SPSS package (Nie *et al*, 1975). Groups were compared using χ^2, Fisher's exact test, and Student's t-test, according to the characteristics of the variable being examined.

Results

Patient characteristics

Nearly half of all the patients received medication *p.r.n.* at least once. There were some important differences between them and their peers who did not. These included age and legal status.

The unit does not accept patients below the age of 18, but there is no upper age limit. The age range of the total patient group was 19–62 years. The *p.r.n.* group was significantly younger than the non-*p.r.n.* group (mean ages 29.5 years v. 33.1 years, respectively, $t = 3.395$, d.f. = 1, $P < 0.05$).

There was no difference between the groups in terms of gender, but only six of the patients in the sample were genetically female (there was also one male-female transexual).

All the patients admitted during the study were compulsorily detained, which is compatible with the policy of the unit that informal patients should be the exception. Not all the patients, however, had been involved in criminal proceedings resulting in admission: 11 were detained under the civil provisions in Part II of the Mental Health Act 1983, and members of this group were significantly more likely to be given medication *p.r.n.* than their peers detained under the criminal provisions in Part III of the Act (Fisher's exact test, $P=0.05$). This is perhaps hardly surprising, as the majority of patients on civil orders had been transferred from general psychiatric units, where they had proved unmanageable. Those admitted under Part III of the 1983 Act had mostly come from prison, where they had been acutely ill but rarely behaviourally unmanageable, or, in a few cases, they had come from special hospitals for rehabilitation.

Most of the patients (75%) had schizophrenia, 12 of these receiving medication *p.r.n.* and 12 not. Of the remainder, the one with affective disorder needed no medication *p.r.n.*, while three of the seven with personality disorders did. Thus there were no significant diagnostic differences between groups. Overall, the 12 schizophrenic patients were administered 114 doses of medication *p.r.n.* (mean number of doses per patient 9.5) and the three with a diagnosis of personality disorder a total of 36 (mean 12.0) (difference not significant). In the schizophrenic group, four patients accounted for a disproportionate number (52%) of all doses.

General context in which medication was given *p.r.n.*

A total of 228 doses of medication were given *p.r.n.*, of which 150 were given on the 73 days randomly selected for detailed study. In the great majority of cases (78%) oral medication was accepted by the patient with little or no persuasion, although 12% were associated with physical constraint. Most of the doses (73%) were given at the staff's initiative, although in just over a quarter of episodes the patient requested medication.

The main nursing reasons for giving medication are shown in Table I. About 25% of doses were given when it was thought that physical aggression was likely, but in a further 24% of cases physical aggression or violence had already occurred. In total there were 42 cases, including two of self-harm, in which aggression was clearly threatened or acted out against an identifiable person. Staff were more commonly victims of personal violence or threat (26 of 42 episodes, 62%). In just 20 of the 150 instances of *p.r.n.* medication had there been actual injury to another person. These were mainly contusions, ranging, for example, from a minor facial bruise caused by one patient slapping another, to more serious bruising with abrasion, again to the face, caused by a patient punching a nurse. There were no cases of more serious injury featuring, for example, lacerations or fractures. There were 18 cases of property damage. These ranged from kicking a medication trolley to wrenching a loud speaker from its wall mounting and throwing it across the room.

TABLE I
Nursing reasons for giving medication p.r.n.

	No. of occasions	*%*
To relieve patient distress	49	32.7
To prevent physical aggression	37	24.7
To contain physical aggression	36	24.0
To contain verbal aggression	13	8.7
To relieve distress caused to others	1	0.6
Other	14	9.3
Total	150	100.0

TABLE II
Nursing interventions attempted directly before and after administration

Interventions (not mutually exclusive)	*No. of occasions used*	*%*
Before		
Talking	111	74.0
Distraction	52	34.7
Going to room	43	28.7
Ignoring behaviour	28	18.7
Relaxation	11	7.3
Total	245	
After		
Talking with nursing staff	97	64.7
Talking with doctor	8	5.3
Talking with duty doctor	7	4.7
Total	112	

In most episodes medication was given *p.r.n.* only after other nursing interventions, often more than one, had been attempted (Table II). If appropriate, antecedents to the episode were discussed with patients following medication, in addition to support and counselling (Table II). The patients were otherwise allowed to be alone or to rest. Hence, *p.r.n.* medication formed part of the overall immediate management only of emergencies; from the perspective of this study it formed the 'filling' of the sandwich of nursing interventions.

It was not standard procedure at the unit on the admission of a new patient to specify *p.r.n.* medication. However, when it was thought appropriate to specify such medication, in most cases it was prescribed flexibly. An example of the instruction on the prescription card would be "haloperidol 5-20 mg orally or intramuscularly not more than six-hourly". In order to evaluate the use by nurses of this facility doses were classified as 'low', 'medium' or 'high' according to whether the nurse used the lowest or highest prescribed dosage or an intermediate one. Nursing staff used the highest available dose on 77 occasions (51%)

TABLE III

Time taken for patient to 'settle' after medication administered p.r.n.

Time	*% of administrations*	*cumulative %*
<5 minutes	6.7	6.7
5–30 minutes	42.6	49.3
30–60 minutes	20.0	69.3
60–180 minutes	10.7	80.0
Did not settle	20.0	100.0

and the lowest on 27 (18%). A choice of dose in the medium–high range ($\chi^2=9.2$, 4 d.f., $P=0.05$) and the intramuscular rather than the oral route ($\chi^2=5.9$, 2 d.f., $P=0.05$) was significantly associated with high levels of patient arousal, as judged by nursing staff, before medication.

Patient response to intervention

Table III gives nursing estimates of the time it took patients to 'settle' following *p.r.n.* medication. It can be seen that in nearly two-thirds of incidents the patient settled within an hour. The chances of further behavioural improvement the same day were small. Interestingly, patients who requested medication themselves were noted to settle no more quickly than those who had been given medication at the nurse's initiative.

Drugs given *p.r.n.* included haloperidol, droperidol, chlorpromazine, thioridazine, sodium amytal, and diazepam. Although our study was not primarily designed to compare the efficacy of these drugs, none showed clear supremacy as regards the speed or efficacy with which the patients settled. Table IV shows the details of change in behaviour after *p.r.n.* medication. In more than half of the incidents agitation, verbal abuse, and threatening violence had all been important problems. Each seemed, within the *p.r.n.* group, to have been significantly reduced within half an hour. Sedative effects within the group were correspondingly significantly increased.

TABLE IV

Changes in behaviour before and after medication given p.r.n.

Patient characteristics	*No. (%) of incidents (n=150)*		χ^2	P
	before	*30 minutes after*[1]		
Agitated	121 (80.6)	64 (46.8)	34.5	<0.001
Verbally abusive	91 (60.6)	37 (27.0)	31.4	<0.001
Threatening violence	82 (54.6)	28 (20.4)	33.9	<0.001
Physically violent	56 (36.6)	10 (7.8)	33.7	<0.001
Sedated	10 (6.6)	31 (20.6)	13.6	<0.001
Other side-effects	3 (2.0)	2 (1.5)	0.1	NS

1. After 13 incidents patient was asleep at 30-minute assessment

Discussion

In a little over three months, 228 doses of medication were given *p.r.n.* to 15 patients in one secure unit. This seems a high figure but it must be remembered that four patients accounted for 119 of these doses, while 17 patients took no *p.r.n.* medication at all. The mean rate of such dosage for the remaining 11 patients was under ten, and two of these 11 patients (3 of 15) were not in receipt of any regularly prescribed medication. Taking these factors into account, together with the knowledge that a substantial proportion of the patients were in a state of acute illness or had proved unmanageable on other units, the total use of *p.r.n.* medication seems less remarkable.

There is no published study with which to compare the rate directly. Cooper *et al* (1983), who found such a low rate of medication used *p.r.n.*, were for example observing patients on ordinary general psychiatry disturbed units, and exclusively women. It is also probably inappropriate to compare in isolation the rate of *p.r.n.* administration of medication without considering other factors, such as the level of regular medication, the use of seclusion, and so forth.

Another contributing factor to higher levels of *p.r.n.* medication in our study, and a possible problem with the study, was the rapid increase in the in-patient population. The unit had been opened for only six months at the time of the study, and was still undergoing a rapid increase in patient population. This is particularly likely to have been important, as the number of patients admitted in a disturbed state was also relatively greater than the number admitted in the residual phases of their condition for more rehabilitative treatment. Six months before our study a pilot survey indicated that the 13 patients in the unit at that time were given only 19 doses of medication *p.r.n.* over two months.

Although there are very few figures published on the subject of *p.r.n.* medication, there is an established literature on violent incidents, with which our study bears comparison. Fottrell (1980) and Cooper *et al* (1983) both found that violent incidents were more common at meal-times and medication-dispensing times. Similarly, in our study, 50% of *p.r.n.* medication was given at these times. Unfortunately, our results do not allow us to establish whether medication was given *p.r.n.* mostly simultaneously with regular medication, or whether there was a short interval. Some studies of violence noted variations in frequency according to the day of the week (Bouras *et al* 1982; Cooper *et al*, 1983), but no such variation was noted in our study. This may be a reflection of the different regime in a secure unit.

A potential criticism of the study is that it covered just one time period, and at a time when staff were aware of the object of the investigation. This effect was, however, minimised by looking at medication given *p.r.n.* on randomly selected days; the nursing staff were unaware in advance which days had been thus selected.

Given that medication was only part of the intervention by staff in many of the crises, it is difficult to estimate the extent of its impact. For the tiny minority of episodes (7%) in which resolution followed in less than five minutes, specific medication effect is impossible. The possibility of a placebo effect remains, but there is also a chance that where there was rapid resolution in behavioural crises after a violent act, a sort of cathartic release of tension had occurred. Perhaps the most powerful influence on resolution, however, was the attention of nursing staff, who helped to 'contain' the patient securely. This is particularly likely given that the relatively high nursing staff levels within the unit allowed interventions to be frequent and sustained. For patients who did not settle at all within the 24 hours of follow-up after the *p.r.n.* medication, the medication can be of little specific value. Thus, in just over a quarter of the cases the *p.r.n.* medication *per se* appeared to have been of almost no use. In the remaining cases, however, it must be argued that it had at least some effect. The time scale of the resolution of the problem is suggestive of a genuine pharmacological response in most cases, and the nature and extent of symptom reduction would similarly be compatible with the nature of the medication generally used.

This was a purely descriptive study focusing principally on one element of overall patient management; further related studies might usefully employ a clinical trial design to evaluate the relative contribution of *p.r.n.* medication and the different nursing strategies used to reduce and control disturbed behaviour. Nevertheless this study does confirm that medication can be used effectively and flexibly by nursing staff as one of these strategies. Prescription which allows the availability of medication in an emergency or on demand in turn allows patients some choice. In just over a quarter of the deliveries of medication studied in detail the patient had requested it. Offering a choice of prescription to nursing staff ensured that at least half the time patients received doses at the lower end of the spectrum, while generally getting the advantage of both symptom reduction and avoidance of violence.

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Case Conference

Changing Interactions Between Bipolar Affective Disorder and Anoxic Brain Damage

M. N. COLLINS and R. R. JACOBSON

A manic–depressive young woman received mild brain damage from anoxia after attempting suicide by hanging. The case conference was held to elucidate whether her subsequent symptoms were functional or organic, and what courses of treatment should be tried in this very refractory case.

Objectives

This is a report of a case conference in neuropsychiatry held at the Institute of Psychiatry on 17 November 1988. The case illustrates the difficulties of distinguishing the relative importance of interacting neurological and psychiatric abnormalities in a patient who has both, but in whom communication is impaired. The conference was asked to help the presenters come to diagnostic and therapeutic conclusions. The case also illustrates the strengths and weaknesses of modern neurological investigative techniques.

Presentation of case

Miss A, an 18-year-old single Caucasian student, was admitted to her catchment area general psychiatric hospital on 13 February 1988, under Section 2 of the Mental Health Act 1983, with mania. Her symptoms had escalated over several days, having been preceded by an episode of depression. Her illness proved difficult to treat despite appropriate use of neuroleptics and lithium. During the month after admission she became severely depressed, then manic again after tricyclic antidepressants were started, and then again depressed. Electroconvulsive therapy (ECT) was commenced, but after the third treatment she attempted suicide by hanging herself from a shower rail with her dressing-gown cord, on 15 March 1988. When found, she was pulseless and cyanosed, with dilated pupils, but she was rapidly resuscitated by an experienced medical team from the general medical wards. The maximum time she had been unobserved was ten minutes, so her anoxia would have been relatively brief.

Following a few days in intensive care, she was taken via a general medical ward to a neurological unit, where she was admitted two months after the hanging. Her persisting problems were mutism, dystonia, dyspraxia, urinary incontinence, and poor self-care. She was assessed for rehabilitation but considered unsuitable because of "manipulative behaviour" (reflecting, we suspect, variability in performance of tasks) and lack of motivation. She was then transferred to a psychiatric unit three months after the hanging attempt. Although psychiatric staff did not observe significant depression, noting normal sleep and appetite, and the absence of diurnal variation in behaviour, they started her on tricyclic antidepressants, without apparent benefit. She was transferred for a neuropsychiatric assessment on 1 September 1988, nearly six months after the hanging, to address the question of therapy in the light of the relative importance of neurological and psychological factors in her presenting condition.

Family history

Her mother, aged 45, has a history of recurrent depression, requiring hospital admission once in the past. A paternal aunt in Ireland has been diagnosed as schizophrenic, and her maternal grandfather is said to have had 'emotional difficulties' after World War II. Her elder sister is depressed and treated with a tricyclic antidepressant, although she has never been admitted, and a younger sister is reported as being depressed with difficulty coping at school.

Both parents are Catholics of Irish origin, but have lived in England since marriage. The father, aged 47, is a buildings officer, who works at the hospital where his daughter hanged herself. Miss A has always been the 'apple of her father's eye', as 'near perfect as a daughter could be'.

Personal history

Birth and developmental milestones were unremarkable, and childhood was happy. She was very intelligent, obtaining 13 'O' levels, and she was studying for 'A' levels with the intention of going to university to read law. She performed badly at interview for the University of Cambridge, was rejected, and became depressed, this being the first hint of psychiatric difficulty. Four months later she was admitted to hospital, as described.

She had no history of drug or alcohol abuse. Premorbidly she was hard-working, conscientious, reasonably outgoing, at times stubborn. She had male friends but no special boyfriend.

Present condition

Miss A was co-operative and able to follow simple commands, although, left to her own devices, she tended to sit or lie and do little. She was episodically incontinent of urine. Every few minutes she grimaced, closing her eyes tight shut and pouting, and often turned her head fully to left or right for a few seconds. She then returned to normal eye contact. Her speech was almost non-existent, with occasional (appropriate) one-word or two-word replies to questions. Her mood was very difficult to assess. Her face was usually expressionless; there were no obvious biological disturbances, nor was she overactive or tearful. Nothing suggested she was having psychotic experiences. Cognition was also hard to assess. On the rare occasions she had answered questions, she had sometimes been orientated in time and place, sometimes not. She knew her birthdate and could write her name and address on request. Memory was otherwise untestable. She had a severe defect in initiative and marked difficulty with dressing, eating and washing.

Physical examination

General examination was normal. In the neurological examination, cranial nerves (as far as these could be tested), muscle tone, power, reflexes, and plantar responses were all normal, as was fundoscopy. She had a movement disorder characterised by pouting movements of the lips, choreiform and dystonic movements of the hands, especially when accompanying normal tasks such as shaking hands. She exhibited forced blinking and had choreiform movements affecting orbicularis oculi bilaterally, suggesting blepharospasm.

Her gait was wide based and accompanied by dystonic arm and hand movements. She had general incoordination. Some movements appeared manneristic. She showed a disorder of visuomotor co-ordination and range-finding such that she could perform the finger–nose test when the examiner's finger was in front of her, but not when pointing laterally; when going through a doorway or sitting in a chair, she felt for the outlines with her hands before passing through or sitting down, as if unable to localise them in space otherwise. The blink reaction to visual threat was absent. Frontal lobe examination was impossible. She exhibited no cortical release reflexes.

Investigations

Routine biochemical and haematological investigations were normal. She had had three electroencephalograms (EEGs). On 28 March 1988 (two weeks after the hanging) there was generalised slowing (2–4 Hz) over both hemispheres, with no response to auditory stimuli, suggesting a prominent anoxic disturbance. On 24 May 1988 the record had improved, and showed irregular activity at 7–8 Hz, with slowing over the right hemisphere. On 30 September 1988 there was rhythmic activity at 9–10 Hz, although often 6–7 Hz, with runs of irregular theta activity in the left temporoparietal area, generalised episodes of sharp waves, and some sharp forms in the right temporal area; this was abnormal.

Computerised tomography (CT) of the brain on 10 July 1988 showed mild prominence of the sulci frontally, and the left Sylvian fissure was wider and deeper than normal. Single-photon emission computerised tomography (SPECT) on 20 September 1988, using technetium-labelled HMPAO, was normal. Magnetic resonance imaging (MRI) on 1 November 1988 was initially reported as normal, but on review was thought possibly to show slight cortical atrophy, especially frontally, and the basal ganglia were darker than normal, suggesting iron or calcium deposition, as may be seen in Hallervorden–Spatz disease.

Interview

An edited video of Miss A, made on 13 October 1988, was shown. Also present were Miss A's mother and a family friend.

DR JACOBSON (*consultant neuropsychiatrist*): Sit down in the middle chair.

Miss A does so, feeling the outlines before sitting in the chair.

DR JACOBSON: Can you tell me how you are?

Miss A looks around, stares, blinks, and grimaces.

DR JACOBSON: Can you point at the camera?

Miss A does so.

DR JACOBSON: Point to your mother.

Miss A does so.

DR JACOBSON: Pick up a pen off the table.

Miss A does nothing. Dr Jacobson repeats the instruction.

Miss A does nothing.

DR JACOBSON: Point to the pen.

Miss A picks up the pen.

DR JACOBSON: Put the pen on the yellow paper.

Miss A turns her head and grimaces. Dr Jacobson repeats the instruction three times. She finally obeys the instruction. Dr Jacobson holds up a piece of paper with the written instruction "Clap your hands". Miss A looks at the paper, sighs, grimaces, looks at her mother, yawns, then eventually whispers "Clap your hands".

DR JACOBSON: Can you put your arms out in front of you?

Miss A does nothing, despite several repetitions of the instruction. Dr Jacobson initiates the motor act by lifting Miss A's left arm. She holds both arms out.

DR JACOBSON: How are you feeling?

Miss A stares vacantly.

DR JACOBSON: Good or bad?

Miss A does not respond.

DR JACOBSON: Can you pick up the small piece of paper and give it to your mother?

Miss A does nothing. Dr Jacobson repeats the instruction, and with no response from Miss A, initiates the required movement. She performs the task once the movement has been initiated.

DR JACOBSON: Can you write your name on the paper?

Miss A writes her name legibly, then adds her address and telephone number.

DR JACOBSON: Can you do up the buttons on your jumper?

Miss A does nothing. Dr Jacobson repeats the instruction several times with no response, then initiates the required movement by placing Miss A's hand on the button. She performs the action well, once the movement has been initiated.

DR JACOBSON: We have finished now; you can get up and go.

Miss A remains seated. Dr Jacobson pushes lightly to initiate the movement. She gets up and walks out of the room.

Progress

While on the ward, considerable variability in Miss A's functional abilities was noted. On some days she did little for herself, requiring assistance to eat, dress, wash, and go to the toilet. On other days she was much more able to do these things for herself. There was no detectable pattern to this. Her speech had always been confined to one-word or two-word utterances, which were infrequent. In view of the possibility of underlying depression, masked by her neurological status, we started treatment with antidepressants. We chose protriptyline because of its potentially alerting properties, and she was able to tolerate maximal doses.

On 20 October 1988, the ward charge nurse reported the observation that Miss A's pupil size exhibited bursts of rapid changes, showing marked dilation and constriction against constant illumination, and that Miss A appeared intermittently not to be paying attention. This observation was confirmed by medical staff. At this point we decided that there was sufficient evidence for a seizure disorder to warrant a trial of anticonvulsants. Until now we had been advised by our neurological colleagues that Miss A had no evidence of seizures. We, however, felt that partial complex seizures of frontal lobe origin could explain some of her symptoms; we therefore prescribed carbamazepine.

Within a week there was a dramatic improvement in her performance. She was much better at eating, dressing, washing, and going to the toilet, and there was virtual resolution of grimacing, pouting, blinking, and head-turning. The pupillary changes were no longer observed. Her speech also improved, with more frequent utterances involving sentences of up to eight words, grammatically and contextually appropriate. All these observations were made as objectively as possible, using a specially designed form. This form broke down aspects of daily living into component parts, allowing a score for each component, depending on the degree of independence of task completion. Thus "Getting up/Washing" had 21 separate components, each of which was scored. The improvement in total scores following the introduction of carbamazepine was obvious and convincing.

This improvement lasted three weeks, but her performance then began to fall off in a very different way. She became overtly depressed, looking sad, crying at times, and saying on more than one occasion "I feel dead". Although her performance on the rating scale deteriorated, there was no return of grimacing, pouting, blinking, head-turning, pupillary changes, or episodic loss of attention; the slight improvement in frequency and quantity of speech was sustained. Most recently she had begun to refuse food and drink.

Conference discussion

DR COLLINS (*lecturer in psychiatry presenting case*): Miss A has a severe bipolar affective disorder which was difficult to treat. As a result of anoxic brain damage she has dementia with some frontal lobe features. She also temporarily had symptoms consistent with ischaemic dysfunction in the watershed zone between the posterior and middle cerebral artery territories, namely the visual range-finding abnormalities. This had been labelled hysterical. She also has dystonia, associated with MRI abnormalities in the basal ganglia.

We are unsure to what extent her current state is 'neurological' or 'psychological'. Although there is a very strong organic 'feel', the investigations show only slight abnormalities. To what extent does a continuing mood disorder affect her performance? Is it possible that the suicide attempt incidentally ameliorated the mood disorder?

We would propose that she has been suffering from partial complex seizures, of frontal lobe origin, possibly for months, and that her dramatic response to carbamazepine reflected treatment of this, with the unfortunate consequence that her mood disorder paradoxically worsened.

DR JANOTA (*consultant neuropathologist*): May I first comment on the MRI scan? I have discussed this already with Dr Hoare [consultant neuroradiologist], who has seen the films. The diagnosis of Hallervorden–Spatz disease is impossible on the basis of the scan alone. In that condition there is iron deposition in the basal ganglia, associated with dystonia. However, the onset is in childhood, and we have heard no evidence of any problems until after the hanging attempt.

DR COLLINS: We too discounted Hallervorden–Spatz disease. I would be interested to know whether the appearances, implying iron or calcium deposition, could result from an anoxic episode eight months previously.

DR JANOTA: The basal ganglia are vulnerable to anoxic damage. I do not know precisely what changes would be expected eight months after an anoxic episode, but it seems likely that anoxia is the cause of the scan findings in this case. I am not dissuaded by the minor abnormalities on the scans from suspecting severe anoxic brain damage in this patient. Anoxic lesions, in my experience, may be widespread, causing severe neurological dysfunction (in this case dementia) without producing much change on CT or MRI scans.

DR ELWES (*senior registrar in neurology*): I agree that the abnormality in the basal ganglia is a result of anoxia. The abnormality correlates with the clinical picture of dystonia. I am not, however, at all persuaded by the arguments for frontal lobe seizures. I think that everything described by you and shown on the video represents dystonia. The EEG does not provide evidence for an ictal disturbance, although EEG telemetry would be a better way of assessing this. If others thought seizures likely, then telemetry might be worth doing, but I do not think you have made a sufficient case.

DR COLLINS: How do you explain the response to carbamazepine?

DR ELWES: As well as being an anticonvulsant, carbamazepine is also used in affective disorders and probably has a non-specific psychotropic effect. More particularly it is used in paroxysmal dystonias in childhood. I think her improvement in praxis represents removal of the impairment her dystonia was causing.

DR JACOBSON (*chairman*): What about the more general question of brain damage?

DR ELWES: I think she has evidence of anoxic brain damage clinically, in the form of dysphasia, dyspraxia, and frontal lobe symptoms such as lack of initiative and urinary incontinence. I am surprised that there is nothing more dramatic on the scans.

DR REVELEY (*consultant psychiatrist*): I am very struck by the severity of her bipolar affective disorder. Am I right in thinking that this was so before the hanging?

DR COLLINS: Yes, this is documented in the case notes. She received very large doses of neuroleptics, lithium, and antidepressants, but remained psychotic and behaviourally disturbed. This was why ECT was started.

DR REVELEY: I think it is possible that affective disturbance is more important than at first appears. Obviously the issue of epilepsy needs to be resolved and I feel that it would be reasonable to perform EEG telemetry. If there is no evidence in support of frontal lobe epilepsy, then I think her depression should be vigorously treated. In the face of failure of tricyclic antidepressants, I would recommend ECT.

PROF. LISHMAN (neuropsychiatrist): As this case unfolded, I thought we were going to hear an account of a predominantly psychiatric disorder, with a finale of successful response to psychiatric treatment. I am therefore surprised at the more 'neurological' direction the presentation has taken, and have to say that I disagree with this emphasis.

I have seen several patients with evidence on scans of severe brain damage who perform much better than Miss A in their speech, praxis, communication, and movement. I believe Miss A has at most mild to moderate brain damage. I am struck that the CT scan might even be passed as normal, although I accept the balance of expert opinion that there is probably mild cortical atrophy. Similarly the MRI scan is remarkable for the lack of abnormality, apart from in the basal ganglia. I think it is most unlikely for her to have severe brain damage without there being more to see on the scans.

When I saw the video, I was struck by Miss A's posture, with slumped shoulders and head bowed forward – the picture of dejection. I think she is severely depressed and that this is the major reason for her severe functional deficits. I would recommend a course of ECT with some urgency, in view of her recent refusal of food and drink.

Regarding Dr Collins' interesting suggestion of a diagnosis of frontal lobe epilepsy, I remain unconvinced. I do not know the significance of varying pupil size in patients with anoxic brain damage, but do not think it provides evidence of seizure activity in the absence of other paroxysmal clinical features. I would also expect evidence of epilepsy on the EEG. I agree with Dr Elwes that she is displaying typical dystonia, and that her beneficial response to carbamazepine does not result from its anticonvulsant action.

If this strategy is not successful, I might consider abreaction.

DR COLLINS: We did in fact perform abreaction on Miss A, although I omitted it from the presentation. Under intravenous diazepam Miss A was able to communicate occasional appropriate 'Yes' 'No' answers. She said her name, but little else. At one point she made a joke, referring to the fact that a nurse had the same first name as she did; when the nurse said "I am called A too", Miss A pointed at a third nurse and, with a smile, said "A three". While under abreaction, there was no grimacing, pouting, or other abnormal movement. No further information was obtained.

PROF LISHMAN: My final suggestion, if ECT fails to produce a good response, is to consider using L-dopa. Some patients with frontal lobe symptoms, especially lack of initiative, may respond to dopaminergic stimulation.

DR JACOBSON: We have considered using bromocriptine, but are worried about possible side-effects, particularly on Miss A's dystonia.

DR BINNIE (*consultant clinical neurophysiologist*) was unable to be present at the conference, but he subsequently watched the video, having noted the comments of other participants in the discussion. He made the following comments:

A very wide range of ictal phenomena can occur in epilepsy of frontal lobe origin, and some of these are so bizarre as to invite a psychiatric explanation, notably bimanual, bipedal seizures with hand-clapping, stamping, rocking, and pelvic thrusting, etc. However, the pouting, grimacing, and blepharospams shown do not fall within the range of bizarre behaviour commonly seen with mesial frontal seizures, and seem more readily explicable as part of this patient's more general movement disorder. I do not know the significance of the rapidly fluctuating pupil size in this context, but it would not appear particularly suggestive of epilepsy. A response to carbamazepine, as seen here, often confuses rather than clarifies the differential diagnosis between epilepsy and a psychiatric disorder, because of its known psychotropic effects. The lack of epileptiform activity in the interictal EEG contributes little to the diagnostic assessment; patients with seizures of mesial frontal origin often exhibit no interictal EEG abnormality and, indeed, ictal changes are often confined to brief runs of generalised slow activity, the significance of which may be overlooked unless simultaneous video recording is performed.

I do not, therefore, think there is evidence that this young woman has epilepsy, but if the matter is sufficiently in doubt, it could be resolved by capturing some of the supposed ictal episodes during EEG and video telemetry.

Summary

Before the conference, the presenters considered that the aetiology of this patient's symptoms was more organic than functional. Although there was some disagreement among the discussants, the conclusion was that we should reconsider the possibility of a strong functional component to several of the symptoms. Our proposal that partial complex seizures of frontal lobe origin might explain some of the symptoms and signs was rejected.

Overall, we feel that this case illustrates the complexity of understanding what is happening where neurological and psychological symptoms coexist, particularly when verbal communication is so limited. The clinical management was extremely difficult, and although the case conference engendered several ideas on how to proceed, clear-cut answers were not immediately apparent, nor did the passage of time clarify the issue (see Addendum).

Addendum

As a result of the conference's recommendations, and because Miss A continued to refuse most food and drink, a course of bilateral ECT was started, comprising ten treatments over five weeks; carbamazepine was stopped, Miss A's mood lifted, and she again began to eat and drink, smile appropriately, and look after herself reasonably well. She hardly spoke at all however, and her dystonia recurred.

While awaiting admission to a neurological rehabilitation unit for physiotherapy and speech therapy, she became disturbed over Christmas and New Year. She became very restless and overactive, pacing and sometimes running around the ward, occasionally leaving the premises.

She slept very little and was at times sexually disinhibited, masturbating openly on the ward. We diagnosed mania. Despite progressively larger doses of neuroleptics (droperidol, up to 240 mg per day) and the addition of lithium carbonate (to a serum level of 1 mmol/l), her mania proved extremely difficult to control. We also tried less established treatments such as the addition of clonazepam, to no great effect.

By February 1989, she was on high doses of psychotropic drugs; self-care was very poor, and she was mute. We felt unable to assess her mental state accurately, and therefore withdrew all medication over two weeks. She then revealed (a) over-activity, minimal sleep, and occasional inappropriate behaviour, (b) relapse of dystonia, and (c) bursts of rapidly varying changes in pupillary size accompanied by a failure to pay attention to others. Reintroduction of carbamazepine and droperidol produced improvement in all three sets of abnormality, although her mood improved to a level no better than that following her course of ECT.

To complicate matters further, Miss A's younger sister became suicidally depressed and was admitted to another hospital. As the anniversary of Miss A's suicide attempt approached, the younger sister became increasingly ill, manifesting near mutism and refusal to eat and drink, with occasional episodes of bizarre posturing; her behaviour closely mirrored that of Miss A.

Presently both sisters remain in-patients, and the entire family has been engaged in family therapy. There has been no significant change in Miss A's condition over several months.

Theoretical note

The literature on the effects of non-fatal hanging is not extensive. Our patient clearly suffered a degree of brain damage, confirmed by the EEG evidence and by the more sophisticated brain scans. However, the degree of functional impairment was not matched by scan abnormalities. Our patient illustrates how difficult it can be to assess the contribution of functional illness to the overall picture in the presence of neurological dysfunction, especially where communication is impaired. Some features of behaviour may be labelled 'hysterical', when they in fact derive from organic disorder. For example, the visual range-finding difficulties, well described by Howard *et al* (1987) as deriving from lesions in the watershed zone between posterior and middle cerebral artery territories, were initially thought to be related to psychiatric disorder.

Partial complex seizures of frontal lobe origin may present with a range of bizarre behaviour (Quesney, 1986; Delgado-Escueta *et al*, 1987). The presenters were impressed by this possibility, and the combination of observations on pupil reactions and response to carbamazepine seemed to provide support to the idea that at least some of Miss A's behaviour might be epileptic activity. However, the conference rejected this suggestion.

Finally, we note that despite sophisticated neuroradiological investigations, there remained uncertainty as to the relative contribution of psychological and neurological factors to the behavioural dysfunction of Miss A.

Acknowledgements

We wish to thank all the discussants for helpful comments on the manuscript, and the nursing staff on Willow Ward, Springfield Hospital, for their invaluable contributions to the management of Miss A.

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Contents: Unresolved grief/Long-stay psychogeriatric patients/Asian patients and the HAD scale/HAD and ROC/CT findings in schizophrenia/Chronic psychoses in Turner's syndrome/Aggressiveness, anxiety and drugs/Auditory hallucinations during oculogyric crises/Reviewing reviewers/Failure to convulse with ECT/Asian suicides/Olfaction and psychiatry/Pathological jealousy and pimozide/Durkheim's views on suicide.

Unresolved grief

SIR: Nightingale (*Journal,* December 1989, **155,** 862–864) described a negative therapeutic reaction occurring as a result of unresolved grief. Each time the patient (Mrs A) experienced symptom relief during a psychodynamic therapy session, she later experienced a return of her depression. Psychodynamic mechanisms are proposed to explain this: in particular, object relations and the concept of 'triumph' over the lost object. 'Triumph', if particularly strong, may interfere with the process of reparation towards a loved object. Each time 'triumph' was experienced her guilt and symptoms returned.

An alternative explanation can be drawn from cognitive-behavioural approaches. Our clinical work with family members who have survived, and often witnessed, the death of a member by homicide reveals similar changes to Mrs A. Survivors are exposed to the severest psychological trauma. Symptoms of post-traumatic stress disorder (DSM–III–R, American Psychiatric Association, 1987) are common. Ruminations, nightmares, constant fear (which can generalise to severe social handicap), and physiological disturbances are also common. Belief in the world as a safe place is destroyed. The trauma results in new cognitive attributions about themselves and the world that can be understood without reference to psychodynamic conflict.

We would like to suggest that other factors, based on goal conflict, could explain the negative therapeutic reaction described. A number of behavioural goal-directed conflicts are readily evident from the brief case description. The ambivalence towards her husband was the result of an approach – avoidance conflict. She felt drawn towards him because he "showed ... the kind of care she never had at home", yet felt repelled because of his impotence. Continuing to accept his caring meant giving up children of her own – clearly a difficult dilemma. This conflict was not resolved when he was alive and would be evoked when he was remembered. Regarding her daughter, she is trapped in an avoidant–avoidant conflict. She wishes to avoid the stress of parenting the child but also wishes to avoid the feeling that she is uncaring. The approval of authority figures ('medical advice') helped resolve the conflict. However, this was overturned by the disapproval of a potentially more powerful figure (modelling theory; Bandura, 1969), a priest (given that she was a "deeply religious woman").

It is likely that this experience led her to a belief that one must be stricken by loss, even of ambivalently regarded objects, if one is to be a good person and obtain the approval of powerful figures. Grief itself becomes an approach – avoidance conflict. To experience relief she must endorse the belief that she is a bad person, which she wishes to avoid. To be a good person she must experience grief, which she also wishes to avoid. The changes seen in therapy ('triumph' and later symptom recurrence) can be viewed as movement between equally desirable but mutually exclusive goal states. Resolution of the conflict likely occurred with a cognitive shift, (explicitly or implicitly reinforced by the therapist – an authority figure), that the previous belief was erroneous. We also suspect that the power of authority figures was enhanced by a set of learned self-attributions that she was inadequate, a result of previous learning within a family that modelled inadequacy (e.g. her father) and failed to provide esteem building feedback ("was not close").

Conditioned responses may also play a part in grief. For example, a knock at the door or a telephone ringing may become associated with the news (or witnessing) of a loss (conditioned emotional reaction). The stimuli elicit avoidant responses, and through generalisation 'avoidance' permeates the survivors life (Amick-McMullan *et al,* 1989). Avoidant reactions may also effect psychotherapeutic endeavours – talking about a loss (and other losses) may elicit avoidance reactions and seeming lack of progress in therapy. Exploring Mrs A's automatic thoughts (Beck *et al*, 1979) when she experienced relief may have helped her uncover dysfunctional thinking.

We believe it is too simplistic to consider only psychodynamic factors. These can be proposed as 'fundamental mechanisms' to understanding. However, they are filtered through the executive and cognitive functions of the ego. Therefore previous learned cognitive attributional sets and conditioned responses must also be considered. All three mechanisms operate together in any case of grief and therapeutic work.

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SIR: Fisher & Murdoch have reformulated the case material I presented (*Journal*, December 1989, **155**, 862–864) and the mechanism of the patient's recovery using explanations drawn from cognitive and learning theory. I am grateful to them for their interest and in most respects I regard their theorising as compatible with my own. However, I would like to take issue over two points where our thinking diverges.

Firstly, they suggest that, as a result of her (earlier) experience of the disapproval of a powerful authority figure (a priest) at her seeming failure to mourn her daughter, Mrs A developed an approach–avoidance conflict regarding her grief whereby she believed that to experience relief she must endorse the belief that she was a bad person, which she wished to avoid. They go on to suggest that resolution of this conflict occurred with a cognitive shift (reinforced by the therapist) that the previous belief was erroneous. This explanation fails to take into account the importance of the patient's guilt over her aggressive feelings towards both her husband and daughter. Exploration of this, in psychodynamic psychotherapy, allowed her to express these feelings to the therapist and to face her fantasy that she was indeed a bad person. Interpretation to her of her *unconscious* fantasy that she was *continuing* to harm her husband (by triumphing over him) each time she experienced relief from her grief, allowed her to free herself from the vicious circle.

Secondly, I would wish to emphasise that the significance of authority figures in this patient's mental life lay in the way in which they interacted with her own internal world, that is with her internal objects. Exploration and interpretation of this was important in helping her to establish a less harsh view of herself and in freeing her from the need for self-punishment.

In summary then, I disagree with Drs Fisher & Murdoch over their failure to consider the unconscious meaning of the patient's symptom pattern and the importance of the interaction between real external figures and the patient's internal objects.

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Long-stay psychogeriatric patients

SIR: Hilton *et al* (*Journal*, December 1989, **155**, 782–786) provide useful cross-sectional data revealing very high levels of dependency among long-stay psychogeriatric patients compared with residents of a local authority home. However, another perspective can be provided by asking whether changes in levels of dependency have occurred over time.

In 1985, Norman, then of the Centre for Policy on Ageing, performed a survey in 14 facilities offering long-stay psychogeriatric care, including our own September ward (Norman, 1987). Dependency was rated using a subsection of the Crichton Royal Behaviour Rating Scale which has been found to correlate well with clinical assessment (Evans *et al*, 1981). The ratings were repeated in December 1989.

In the 1985 survey of our ward, before it was fully open, 15 patients attained a mean score of 8.5 (range 5–11). One patient scored the maximum dependency score (11). Furthermore, Norman reported that 'several' had difficulties with speech, no-one was totally chair-bound and four (27%) required physical help with feeding. The mean score in December 1989 for 18 patients (the maximum number of beds was 20 but two deaths had occurred just before the ratings) was 9.6 (again, range 5–11). However, this time, eight patients scored the maximum of 11, only two had any conversant language, eight were chairbound and 12 (67%) needed help with feeding. Although not documented, our nurses were convinced that disturbance as well as dependence had increased since 1985.

These changes were not merely the effects of a new unit opening with 'easy' patients who subsequently all grew frail together, since only two patients survived from the 1985 cohort. Like Dr Hilton *et al*, our colleagues also believe that their local authority homes have been taking ever more dependent clients. It may be then that this trend, if it is indeed a widespread phenomenon, has led to all but the most behaviourally difficult demented individuals being placed in residential homes. The hospital services then have to cope with an ever more disturbed and disturbing group that cannot (and perhaps should not, given the skilled interventions required) be placed elsewhere.

Secular trends are clearly important for planning, and it would be interesting to know whether or not our experience is purely local. If not, then it must add to the concerns raised by Dr Hilton *et al* concerning the closure of hospital beds for demented patients. The White Paper *Caring for People* (HMSO, 1989) states that "there will be others, in particular elderly and seriously mentally-ill people ....whose combination of health and social care needs is best met by care in a hospital setting. There will be a continuing need for this form of care". Doctors with a proper training in old-age psychiatry develop skills in managing these patients and in supporting those who care for them, both nurses and involved relatives. Wholesale re-location into the 'community', away from specialist supervision, for reasons of financial expediency could lead to deteriorating standards of care for these vulnerable patients.

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SIR: We were very interested to read the report by Hilton *et al* (*Journal*, December 1989, **155**, 782–786) since it is indeed the case, as the authors state, that there is a paucity of literature in this area.

We are able to confirm Dr Hilton *et al*'s results since we have recently completed a detailed observational study of two long-stay psychogeriatric wards which included the Clifton Assessment Procedures for the Elderly Behaviour Rating Scale (CAPEBRS) (Pattie & Gilleard, 1979). We also obtained scores typically higher than those reported by Pattie & Gilleard for a sample of long-stay psychogeriatric patients. Since the long-term aim of our research is facilitating collaborative ventures with neuroscientists, we also collected in-depth observational and reliability data. From this we make the following points.

We found the social disturbance scale of the CAPEBRS to be unreliable when rated by nursing staff, in contrast to Pattie & Gilleard. Dr Hilton *et al* report that the CAPEBRS was completed by the researcher, which may make this subscale more suspect. We feel this is an important point since it is this subscale which details objectionable behaviours so frequently reported in the literature. This unreliability may be due to the low frequency with which such behaviours occur in this setting, which is supported by Dr Hilton *et al*'s finding of an average score of 2.42 from a possible 10 on this subscale, and further supported by our own observational data.

We would suggest that the picture emerging of long-stay psychogeriatric patients is one of gross physical incapacity and dependency and agree with Dr Hilton *et al* on the need for appropriate staff support. However, the role of disturbed behaviour, typically wandering and aggression (e.g. Mann *et al*, 1984) is in our opinion less important in this setting than in earlier stages of the disease process.

Finally, we take issue with Dr Hilton *et al*'s last assertion, that the problems they have identified may describe any London borough. This may or may not be true, but is likely to depend on local availability of resources and, in the light of our comments above, we fail to see why this should make the authors wonder about the appropriateness of general plans for provision in the community.

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Asian patients and the HAD scale

Sir: Surely Chaturvedi is rather too severe in his criticism (*Journal*, January 1990, **156**, 133) of Nayani's translation of the HAD scale into Urdu (*Journal*, October 1989, **155**, 545–547)? He may be right that some researchers: "... have the impression that mere translation of an instrument is sufficient to make it applicable for use in populations of different ethnic or linguistic backgrounds", but a careful reading of Dr Nayani's report shows that he is not one of them. His research intention was not to *use* the scale to measure an Asian population but to *determine its usefulness* (i.e. to find out to what extent it could be applicable and what changes it might need), addressing, in fact, those very issues which Dr Chaturvedi thought were being ignored. Moreover, it is clear that Dr Nayani understands the limitations of word-for-word translation. Various authorities, he says, "... have emphasised the importance of translation of the concept rather than the literal translation of sentences. The HAD was translated on this principle...".

Dr Chaturvedi has opened a can of worms. Probably everyone would agree that if we take rating scales that are validated in one culture only, and use them in other cultures without modification, we can obtain nice neat columns of figures which don't mean anything. On the other hand, if we use different measuring scales, each one culturally appropriate and valid in its place of origin, the results will be more ethnographically satisfying and probably more clinically useful. The snag is that we won't be able to use those results for inter-group or international comparisons; and epidemiology is important.

How can we escape from this dilemma? The usual compromise seems to be to start with a well-known rating scale and translate it, then twist and bend it a bit, knocking off a few apparent irrelevances and substituting one or two 'cultural' features, and hope for the best. Is this right? Is there a better way? If compromises are in order, are there some general rules or principles? How many changes can be made to a rating scale before it becomes a different scale? Any? Of course, a scale taken out of its context should be revalidated; but what does that mean – recalibration against a local clinically-selected reference sample, or something more than that? Are there differences (in this respect) between instruments which identify diagnostic categories, and instruments used only within an agreed category to quantify severity or measure change over time?

If those who are wise in such matters could offer some guidance, I am sure the rest of us would be grateful.

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HAD and ROC

Sir: Razavi *et al* (*Journal*, January 1990, **156**, 78–93) investigate the characteristics of HAD scale in cancer patients. We have some observations concerning the reporting of such research findings.

Firstly, the HAD scale was devised in order to provide clinicians and researchers with estimates of the presence and severity of two separate emotional disorders: anxiety and depression. It was not devised in order to provide a 'global' concept of the presence of psychiatric disorder as does the General Health Questionnaire. There have been several instances of research reports based upon summation of the two subscale scores of the HAD, but this should not be done. Dr Razavi *et al* later present validation for the two subscales separately, and find the performance of the anxiety scale to be relatively poor; this is to be expected when the gold-standard for HAD *anxiety* is the presence or absence of *depression* (with or without adjustment disorder).

Secondly, the purpose of a receiver operating characteristic (ROC) analysis is to illustrate the relationship between false positives and false negatives at different cut-off points on the scale. ROC analyses are analagous to bar-charts – they should convey information more succinctly than the equivalent table. It is the scale points themselves, not the smoothed-out curve, which the reader wishes to examine, in order to judge relative merits of different cut-offs.

The authors state that "the optimal cut-off for the screening of major depressive disorders seems to be 19". This is incorrect. The purpose of displaying the relationship between true positives and false positives is to allow a *choice* of cut-off. The decision will depend on: (a) the prevalence of the target disorder in the study population; (b) the value and feasibility of intervention with cases identified; and (c) the fate which befalls those patients assigned to the wrong category.

An increasing number of reports of psychometric test data are being presented in terms of ROC analysis. As noted above, one purpose of the ROC chart is to enable a suitable cut-off point to be discerned; presenting a series of straight lines joining the points rather than an artificially produced 'curve' is more helpful in this case. A second purpose is to judge the relative merits of a series of tests and, in this case, the judgment rests upon which of the series of 'curves' lies nearest to the top left hand corner or alternatively, has the largest area under the curve. In this case, therefore, smooth curves may suitably illustrate the findings.

Readers requiring further information concerning ROC analysis will find the study by Murphy *et al* (1987) to be useful.

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CT findings in schizophrenia

Sir: I would like to reply to comments by Ingraham and Crichton & Hughes (*Journal*, March 1990, **156**, 444–453) on our recent paper (*Journal*, October 1989, **155**, 444–450).

In our study, multivariate analyses were used in order to correct for age and gender because enough matched controls were not available. In these multiple discriminant analyses, the enlargement of lateral ventricle: brain ratio (VBR_1), although present, was not significant to distinguish schizophrenic patients from normal controls, nor familial patients from non-familial ones. We recently reproduced these computerised tomography (CT) findings in a magnetic resonance imaging (MRI) study (Uematsu & Kaiya, 1988, 1989) of 40 schizophrenic patients and 17 normal controls. The subjects were all males, aged under 50, and there was little overlap with those in the CT study. Student *t*-tests showed significantly higher VBR_1 in schizophrenic patients than normal controls, and in schizophrenics with horizontal transmission than non-familial patients (unpublished data). However, multiple discriminant analyses again showed that, here again, VBR_1 was not a central finding for diagnosis and heredity although VBR_3 was significant both for the diagnosis of schizophrenia and for the differentiation of familial and non-familial patients.

The idea to divide schizophrenics into three subgroups stemmed from the contagion hypothesis (Crow & Done, 1986) related to the retrovirus/transposon hypothesis (Crow, 1984). Crow & Done (1986) found a correlation of age of onset between siblings and a tendency for the disease to occur at an earlier age in the younger sibling. One of their explanations for this finding was that the disease is transmitted from those who already have it to relatives who possess the genetic predisposition. We performed our study based on this hypothesis and showed a possibility that schizophrenia with horizontal transmission is a distinctive disease.

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Chronic psychoses in Turner's syndrome

Sir: Bamrah & MacKay (*Journal*, December 1989, **155**, 857–859) presented a case of chronic psychosis in Turner's syndrome and reviewed the literature. I would like to add the following information to their report.

Firstly, the 45, XO sex chromosome karyotype makes up 51% of Turner's syndrome (TS) while mosaics of the 45 XO/46 XX sex chromosome karyotype make up 18% and 25% carry an X chromosome abnormal in structure (Fishbain & Vilasuso, 1981). Buccal smear analysis will not necessarily identify a mosaic for which karyotypic analysis in leucocyte culture is required (Akesson & Olanders, 1969). Drs Bamrah & MacKay did not specify how their patient's karyotypic pattern was determined. If only buccal smear analysis was utilised, it is possible that their patient was a TS mosaic.

Secondly, Drs Bamrah & MacKay did not make it clear if they restricted their review to psychoses associated with all karyotypic types of TS or to 45 XO only. In addition to the seven cases they found in the literature, I am aware of three others: Beumont & Mayou (1971); Kolb & Heaton (1975); and Money & Mittenthal (1970). Two of these cases were mosaics and may not have been included for that reason.

Thirdly, the literature also contains four cases of TS associated with affective disorder of psychotic proportions: psychotic depressive reaction, endogenous depression and two cases of manic-depressive illness (Fishbain & Vilasuso, 1981). Some of these cases were also mosaics.

Finally, if psychotic reactions are uncommon among TS patients, then the prevalence of TS within schizophrenic females or females in mental institutions should be lower than that in a pool of newborn girls, where the prevalence of negative sex chromation is 0.05% (Akesson & Olanders, 1969). Two studies found *no* TS in large numbers of mental hospitals (MacLean *et al*, 1968) or chronic psychotic females (Anders *et al*, 1968). Another two studies (Akesson & Olanders, 1969; Kaplan & Cotton, 1968) found 0.03% and 0.4% prevalence of TS among mental hospital and schizophrenic women respectively. The one TS patient identified by Akesson & Olanders was an XO while all three identified by Kaplan & Cotton were mosaics. These studies indirectly support Drs Bamrah & MacKay's contention that the "absence of an X chromosome would confer some immunity from major psychiatric illness". However, it appears that immunity may not be conferred on a TS mosaic. This is the reason why the issue of mosaicism is important to this research area.

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Aggressiveness, anxiety and drugs

SIR: The brief article by Kirov on this topic (*Journal*, December 1989, **155**, 846) draws attention to a link between aggressive behaviour and anxiety, and derives a general principle that anxiolytic drugs may be expected to have an anti-aggressive effect. An early review of the effect of drugs on violent behaviour (Goldstein, 1974) found little encouragement for the use of anxiolytic or tranquillising drugs. I have had the opportunity to review more recent literature (Conacher, 1988) in which favourable results have been reported for some classes of drugs, that are not, however, all noted to possess a direct anxiolytic effect. A hypothesis has been advanced that the effect of these drugs is mediated through a serotoninergic system (Editorial, 1987).

Paradoxical reactions to tranquillisers have long been recognised, but there is too little known about these to confidently assert that they arise out of 'an abnormal terrain' such as a previously damaged central nervous system. In institutional environments where crowded conditions prevail, benzodiazepines should probably be regarded as contra-indicated in the treatment of aggression. Clinical experience supports empirical evidence that other drugs can be more effective in selected cases.

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Auditory hallucinations during oculogyric crises

SIR: Auditory hallucinations during oculogyric crises, reported by Chiu & Rogers (*Journal*, July 1989, **155**, 110–113 and October 1989, **155**, 569–570), have been attributed to a temporary imbalance between cholinergic and dopaminergic activity (Leigh *et al*, 1987). This is supported by ocular movements under dopaminergic control (Rascol *et al*, 1989) benefiting from either anticholinergic or dopamine receptor blocking drugs (FitzGerald & Jankovic, 1989).

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Reviewing reviewers

Sir: I have read Ellenberger's book *The Discovery of the Unconscious: The History and Evolution of Dynamic Psychiatry* from cover to cover. Twice. Does this qualify me for some sort of record?

I was quite disappointed with MacDiarmid's reconsideration of the book (*Journal*, January 1990, **156**, 135–139), and taken aback that he considered it acceptable to admit he had not even read it all. I had thought that a *sine qua non* of reviewing was that the reviewer read the piece under review, be it never so long or tedious. It *is* long, but in my opinion not a page too long.

Perhaps because MacDiarmid is not familiar with the whole of the book, the impression I received from his review was not the same as that which I got from the book itself. Ellenberger's chapter on Janet is actually considerably longer than his chapter on Freud, yet approximately half of MacDiarmid's review deals directly with the latter.

I had found that this was one of the strengths of Ellenberger's book that he, as it were, put Freud into perspective, so that one could see what came before, after and at the same time, despite Freud's subsequent and now challenged pre-eminence. I don't think that this is reflected in MacDiarmid's review.

It may seem impertinent, but I don't think it unreasonable to request that people who review books should take the time to read them fully.

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Failure to convulse with ECT

Sir: Failure to convulse with electroconvulsive therapy (ECT) has been the subject of much recent discussion in your *Journal* (*Journal*, January 1988, **152**, 134–136; *Journal*, April 1988, **152**, 571; *Journal*, May 1988, **152**, 712–713). Reference has been made to a number of measures adopted to address this problem including vigorous pre-oxygenation, caffeine priming, reduction in methohexitone dosage, the use of chlorpromazine, cessation of benzodiazepines, the conversion from unilateral to bilateral electrode placement and the introduction of 'high energy' ECT machines. From a quality assurance perspective, it is disconcerting but important to reflect on the range of clinical activities derived from such measures. For this purpose, the psychiatrist's activities can be dissected, temporally, as follows: (a) what is done at the patient's bedside if there is no convulsion on application of the electrical stimulus; (b) what changes, if any, are made prior to the next ECT treatment session.

To the best of my knowledge, neither routine has been surveyed. Discussion with a number of colleagues working in different institutions suggests that an alarming variety of practices and protocols abound. To expound on (a), for example, some psychiatrists will not deliver a further stimulus if a patient does not convulse with the initial one. Other psychiatrists decide to give one, two or three further stimuli before either effecting a seizure or abandoning ECT for the day. Some administer a repeat stimulus immediately after failure of a preceding one. Others ensure that a designated period of time (usually up to a minute) elapses between administrations. Some psychiatrists will not change the original electrical settings for repeat stimuli. Others increase the duration but not the amplitude of the current. Others increase the amplitude but not the duration. Still others increase both the amplitude and duration. Increments in such parameters depend partly on the nature of the ECT machine but are, in any case, often randomly chosen. Some psychiatrists retain the initial electrode position, perhaps exerting more pressure on the patient's skin. Some convert from unilateral to bilateral placement. Many psychiatrists are hesitant or inconsistent in their routine. A familiar scenario may emerge – an electrical stimulus is delivered, a clinical fit doesn't ensue, the anaesthetist and psychiatrist look expectantly at one another, the attendant nurses look politely at the floor.

The described diversity of practice is, I suspect, not restricted to the Antipodes. It is lamentable. The administration of an electrical current to the head is not without morbidity and detractors. There is a need for a more formal survey and more uniform protocol, validated by empirical data. Of course the argument that efficacious electroencephalograph (EEG) seizure activity can occur in the absence of a clinically detectable fit impacts upon this subject and warrants further study. Nevertheless, in the interim, specific attention should be given to whether the application of a repeat stimulus significantly increases the likelihood of a fit and, if the stimulus is repeated, what its optimal characteristics (e.g. amplitude and duration of current) and 'climate' (e.g. degree of oxygenation of patient, concurrent drug therapy) should be.

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Asian suicides

SIR: Soni Raleigh *et al* (*Journal*, January 1990, **156**, 45–50) report that, among migrants from the Indian subcontinent, suicide accounts for 1.8% of male and 2.7% of female deaths. Since 1976, admission of Asians to the West Midlands Poisons Unit following deliberate self-poisoning has increased two-fold for men and four-fold for women. Using local Labour Force Survey statistics supplied by the Department of Employment, we estimate that one in 65 Asian women aged between 16 and 19 years is admitted to hospital following an overdose each year.

Thirty-one Asian suicides occurred in the city of Birmingham between 1984 and 1988. Like Dr Soni Raleigh *et al*, we found most cases were aged under 35. Two of the 17 male suicides and six of the 14 female suicides were by burning. Although only four males and two females died as a result of self-poisoning, seven males and seven females had documented previous suicide attempts, most being drug overdoses.

Risk factors for suicide following suicide attempts by Whites should not be applied to Asians. Among Whites, such factors include being middle aged to elderly, male, socially isolated and clinically depressed, but in the Indian subcontinent most suicides are young people with family problems who do not suffer from psychiatric illness (Merrill, 1989). The epidemiological findings of Dr Soni Raleigh *et al* and our preliminary results from Birmingham suggest that the characteristics of suicide by Asians in Britain are similar to those in the Indian subcontinent. Factors implicated in both suicide and attempted suicide among Asians are therefore similar (Merrill & Owens, 1986).

We have recently completed a clinical study of attempted suicide by Asian women which suggests that they may be at high-risk of future suicide. Compared to Whites, Asian women scored significantly higher on Beck's Suicide Intent (SIS) and Hopelessness Scales (HS) with those whose marital status was 'separated' recording especially high scores. A measurement of traditionalism and intergenerational conflict of values, the Traditional Values Scale (TVS) (available on request) was also administered. A significant positive correlation was found for non-traditionalism, conflict, SIS and HS scores.

We also administered the General Health Questionnaire (GHQ), HS and TVS to all fifth-form students at two Birmingham schools. A response of 'definitely have' to the GHQ questions 'have you recently thought of the possibility that you might do away with yourself?' and 'found that the idea of taking your own life kept coming into your head?', or a response of 'much more than usual' to 'have you recently found yourself wishing you were dead and away from it all?' was made by 24 of 72 Asian females (33.3%) and 16 of 78 Asian males (20.5%), but only three of 52 white females (5.8%) and one of 51 white males (2.0%). GHQ and HS scores were significantly higher for Asian females than other groups and were highly correlated with scores of non-traditionalism and conflict on the TVS.

Suicidal ideation, attempted suicide and suicide are much more common among young Asian women than young white women. Holding non-traditional values and being in conflict with parents are associated with suicidal ideation and, in those who have made suicide attempts, with high suicidal intent. Dr Soni Raleigh *et al* reported an increased incidence of suicide in young married Asian women and we found those who are married but separated and make suicide attempts have high suicidal intent. Although most Asian marriages are 'arranged', we do not believe that arranged marriages *per se* are a major cause of suicide or suicide attempts. Cultural expectations of newly-married Asian women tend towards conformity to prescribed roles. Those with scant regard for traditional values may consider, attempt or succeed in killing themselves.

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Olfaction and psychiatry

SIR: Harrison & Pearson (*Journal*, December 1989, **155**, 822–828) point out that significant olfactory deficits are identified in various neuropsychiatric conditions. Our work on olfactory functions in neuropsychiatric disorders supports this.

We observed that patients with right temporal lobe lesions find it difficult to match identical smells of non-volatile aromatic liquids. This prompted us to develop and standardise a test to differentiate right temporal lobe lesions. This smell-matching test is useful in identifying right temporal lobe lesions, with a sensitivity of about 75% and specificity of about 85% (Abraham & Malthai, 1983; Abraham, 1983). We also found that patients with schizophrenia and affective disorders have lower scores on this test compared with normals, whereas patients with neurotic disorders have normal scores (Sreenivasan *et al*, 1987).

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Pathological jealousy and pimozide

SIR: Byrne & Yatham (*Journal*, August 1989, **155**, 249–251) discuss the case of a heavy drinker suffering from pathological jealousy who responded satisfactorily to treatment with pimozide. Cohen (*Journal*, November 1989, **155**, 714) rightly questions the validity of this case, pointing out that pathological jealousy often improves in alcoholics when they stop drinking. Therefore, it is very difficult to know whether or not the improvement in the patient of Drs Byrne & Yatham was due solely to the pimozide. Nevertheless, we believe pimozide can be used to treat pathological jealousy, and report two cases to demonstrate this.

Case 1: A 52-year-old woman had had an eight year history of systematised pathological jealousy. She claimed that her husband had an eight-year-old daughter by a workmate who was his lover. As irrefutable proof of this she cited the receipt from a postal order on which the street name 'Maria del Valle' appeared. According to the patient, it was obvious that her husband's lover was called Maria del Valle. She did not suffer from hallucinations, showed no other psychiatric symptoms and had no history of psychiatric disorder or alcoholism. A physical examination and an electroencephalograph (EEG) proved to be normal. Pimozide (8 mg/day) was prescribed and there was a slight improvement after one week. After two months of treatment, the patient had stopped talking spontaneously about her husband's lover and daughter. Six months later, she believed that her husband had stopped seeing his lover and said that she had forgiven him for what he had done, although she couldn't love him as much as she had before. Nevertheless, she was still convinced that the lover and the daughter had existed. Now (a year and a half later) the patient is still being treated with pimozide (2 mg/day) At present, she believes that her husband is no longer unfaithful to her but has been in the past.

Case 2: A 56-year-old woman has been suffering from pathological jealousy for 13 years. Symptoms first appeared after she found blood stains on her husband's underwear For her, this was proof that her husband was being unfaithful. She did not suffer from hallucinations, alcoholism or any other psychiatric problem. However, her medical records showed she had a history of high blood pressure, diabetes mellitus and coronary artery disease. She had also suffered from post-partum depression. Pimozide (8 mg/day) was prescribed. After two months of treatment, her family found that she was much calmer and had virtually stopped talking about her husband's unfaithfulness. After four months, she believed that her husband was sorry for what he had done and was more attentive She said he was no longer unfaithful to her, but believed that he had been over the past years. Six months later this improvement was maintained with pimozide (2 mg/day).

In both cases the response to treatment was good, especially if the chronicity of the illness is taken into account.

Finally, we would like to point out the similarity of the two cases in that both patients came to believe that the problem no longer existed but had in the past. This is the probable evolution of a monodelusional psychosis when treated with antipsychotics.

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Durkheim's views on suicide

SIR: Berrios & Mohanna's (*Journal*, January 1990, **156**, 1–9) criticism of Durkheim's views on suicide was biased. Durkheim was writing a sociological theory for suicide and not a psychiatric text. His theory on suicide is now known world-wide in spite of his absence as a contributor in the symposium of the British Medical Association of 1899. His masterpiece, 'Le Suicide' (1897) whether you read the translation in English, Spanish or Arabic (as I did) is clear and did not show any inaccuracy. Indeed, you will find that Durkheim is remarkably modern and scientific in his thinking.

Medicalisation of suicide and acceptance of the absolutists views saturates the environment of suicide with ready-made psychiatric diagnosis for practical and political reasons, and offers a therapy without knowing whether it has any value. Whether the suicide rate is influenced much by the efforts of psychiatrists remains an open question. Because we fail to discover an anti-suicidal substance, we are left with a dash mix of medical and social theories of mental illness and suicide.

Most psychiatrists accept the high risk of suicide and parasuicide in the unemployed. If we apply Durkheim's views, the risk could be reduced by return to employment but if we reject them, the result is abuse of chemicals and mishandling of human experiences under scientific claims. Even the World Health Organization (1968) accepts that 'measures against social isolation and furtherance of satisfying social relations within and outside the family appear to provide the best effective prophylaxis against suicidal tendencies'.

Finally, it may be important and interesting to put the historical record straight. It seems to me that when Durkheim wrote his theory of suicide and alienation, he was influenced much by the writing of the Arabic scholar, IBn Khaldun (1332–1406) and his central concept of social cohesion. A complete French translation of IBn Khaldun's work appeared in the 1860s.

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A HUNDRED YEARS AGO

Rich lunatics and poor

The vacancy in the clinical professorship at the State Asylum in Vienna, caused by the superannuation of the late Dr Leidesdorf, was filled by the appointment of Dr E. von Krafft-Ebing, of Graz, who is already well known by his handbooks on psychiatric medicine and jurisprudence, who entered on the duties of his chair at the commencement of the present winter session. Judging from a report of his inaugural address, which recently appeared in the papers, he made some startling, if not alarming, statements. First of all, he said that in civilised countries 1 in every 200 of the adult population was insane. Griesinger, with doubtless imperfect statistics, gave the proportion for Austria as 1 to 500, and Dr Hack Tuke, after making liberal allowances for the cases not placed under certificates, came to the conclusion that for England and Wales the proportion was 1 to 300. It may be doubted whether Austria is worse off than we are.

The next part of Professor von Krafft-Ebing's statement was that the average of cures among the lower classes was 56%, whereas in the higher classes it was only 16%; and this he attributed to the fact that rich people, when they began to feel mental disturbance, had recourse to hydropathic treatment and other mere palliatives, and were not placed in asylums till too late.

It is well to compare the returns of the English Commissioners in Lunacy with the above. In the report for 1888 we find that the average percentage of cures on admissions was 40.16 in county and borough asylums, 47.24 in registered hospitals, 35.11 in metropolitan licensed houses, 36.44 in provincial licensed houses, and 14.96 of cases in single private care.

From these returns it would appear that our large asylums are far behind Austria in the percentage of cures, 56% being discharged cured in Austria, as against 40% in England. We cannot explain this discrepancy, but would suggest that the asylums of the two countries differ very considerably in their relative proportion to the population, and that, as a consequence, only the more acute, and presumably more curable, pass at once into asylums in Austria. As to the small proportion of the richer patients who are cured, we can only compare the 14.96% of patients in single care in England with the 16% of private cases cured among the Austrian higher classes.

In English private asylums the Commissioners' returns point to a good proportion of cures; but those who know the inner working of private asylums know that a very large number of patients are sent to private asylums only after every other form of treatment has failed, and therefore the chances of their recovery are less. This seems, according to Dr von Krafft-Ebing, to be still more the case in Austria; and we suppose that on the Continent, hydrotherapy, at least baths and *Kurhäusen*, are much more commonly made use of for mental patients than with us. That there is such a difference between the cases among the richer and the poorer classes is by no means made out; all those richer patients, who are cured by hydrotherapy, travel, and the like do not come into the calculation at all – so that, in fact, the argument might rather be that general forms of treatment are so well carried out in Austria that only the incurable cases settle naturally into asylums. It may be said that surely equally careful and skilled general treatment is given to the richer English patients as to the Austrians, but this is not quite so clear. We believe that, though insanity is beginning to be seriously studied in England, hitherto foreign physicians have had a more general training in psychiatric medicine than have their English brothers. At the Vienna asylum the resident staff is much larger than that of an English asylum of the same size, and the students are more numerous and are more carefully taught; so that we should be more inclined, as we said before, to attribute the small returns of cures among the upper classes in Austria to the nature of the cases admitted than to the antecedent treatment.

Dr von Krafft-Ebing speaks of the future of hypnotism in the treatment of insanity with some hope. We can only say that in England the results of trial have been very unsatisfactory.

Reference

British Medical Journal, 9 November 1889, 1047–1048.

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British Journal of Psychiatry (1990), **156**, 752–754

Reading About . . .

Borderline States

A new diagnostic category, borderline personality disorder, was included in the psychiatric classificatory system for the first time when it appeared in the DSM–III (American Psychiatric Association, 1980). However, the label, some of the associated concepts, and of course, the patients, have all been around for much longer. The contemporary concepts have two roots. The first is in the psychoanalytic literature: on the borderline between neurosis and psychosis. These patients are chronically unstable and impulsive, have difficulties in close relationships, and are prone to brief psychotic episodes. The second root is in the general psychiatric literature: on the border of schizophrenia. These patients are cold and aloof, with odd speech, depersonalisation and derealisation, and recurrent illusions. This dual origin will be reflected in the choice of papers and books.

Within the psychoanalytic literature, a good starting point is the paper on psychoanalytic investigation of and therapy in the borderline group of neuroses (Stern, 1938). Stern describes the vulnerability and fragility of such patients, and points out some of the pitfalls that await the therapist. From Stern one might move on to *The Ailment* (Main, 1957), an excellent account of what it is like to work with such patients at an in-patient unit.

Although the British object relations theorists (Balint, Winnicott, Fairbairn, and Guntrip) did not appear to use the term 'borderline' in a diagnostic sense, they made an important contribution to this area, by providing a new theoretical and conceptual framework within which such patients might be understood. Their contribution is well summarised by Sutherland (1980), although some readers might prefer to look at original texts, such as Balint (1960) (*The Basic Fault*), Winnicott (1965) (on the false self), and Fairbairn (1940) (on the schizoid personality). Segal (1975) provides a good general summary of Kleinian theory, and Rey (1979) and Rosenfeld (1979) give more specific accounts on the borderline patient from a Kleinian perspective. In particular, Rey describes the patient's 'agoraphobic–claustrophobic' anxieties.

Kernberg has made the most comprehensive effort to describe and define a particular psychology of the borderline personality, which he calls 'borderline personality organisation'. This is defined in terms of the patients characteristic defences (splitting and projective identification), reality testing (better than for psychotic patients), and identity diffusion (worse than for neurotic patients). His most important early papers are contained in *Borderline Conditions and Pathological Narcissism* (Kernberg, 1975), but *Internal World and External Reality* (Kernberg, 1980) is much easier to read! The adventurous reader might also like to grapple with Kernberg's rival, Kohut (1977), one of the leading exponents of self-psychology. Shapiro (1978) has written a good summary of the psychodynamics of the borderline patient. Rinsley (1984) compares borderline and narcissistic conditions in a paper which is short and to the point.

Moving back to this side of the Atlantic, Steiner (1979) is a British writer who has applied Kleinian theory to such patients, and who has more recently begun to talk of a specific pathological organisation (although not a 'borderline position'), in relation to the more widely known paranoid–schizoid and depressive positions (Steiner, 1987).

At this stage, one might profitably go right back to some of Freud's fascinating and eminently readable original case studies on hysteria. The reader might like to consider which, if any, of Anna O (Breuer), Emmy von N, Lucy R, Katharina, or Elisabeth von R, the original hysterics, would today be described as borderline (Freud, 1985).

Turning now to the phenomenological literature, Stone (1980) remains the best and most readable of comprehensive guides to this area. The paper by Zilboorg (1941) on ambulatory schizophrenia is interesting, as is the work on pseudoneurotic schizophrenia (Hoch & Cattell, 1959). The relevant chapters in *The Transmission of Schizophrenia* (Rosenthal & Kety, 1968) give some useful background information.

Of the more modern papers in the mainstream psychiatric literature, Spitzer *et al* (1979) is a must.

This gives an account of how a partition was developed between schizotypal and borderline personality disorders, and how the DSM–III criteria for each of these two new diagnostic categories were finalised. Spitzer & Endicott (1979) provide more information on this subject, and on the final choice of the term 'borderline'.

There is a whole series of papers on the development of the Diagnostic Interview for Borderlines (DIB), still the best of its kind. One should start with Gunderson & Singer (1975), followed by Gunderson & Kolb (1978) and Gunderson *et al* (1981). Barrash *et al* (1983) applied cluster-analytic techniques, with some success, to the DIB criteria.

Pope *et al* (1983) provide information on the validity of the DSM–III diagnosis, and McGlashan (1986) gives interesting follow-up data. The study by Kroll *et al* (1982) is one of the few empirical works carried out in the UK.

For those interested in the validity of the partition between borderline and schizotypal, and the respective relationship of each of these to schizophrenia, Kendler *et al* (1981) and Gunderson *et al* (1983) re-examined the Danish adoption study records, finding evidence to suggest that: (a) the partition is valid (although the DSM–III criteria for schizotypal personality should be refined); and (b) schizotypals are related to schizophrenics, borderlines are not. Torgersen (1984) showed that monozygotic twins of schizotypal probands are often schizotypal and never borderline.

Some writers (Stone, 1980; Akiskal, 1981) have argued that the border in question is not with schizophrenia, but with depression. The literature on the relationship between borderline personality and affective illness has been thoroughly reviewed by Gunderson & Eliot (1985).

The reader might like to compare changing views on the validity of the diagnosis before and after the DSM–III and DIB came into use. Liebowitz (1979) and Tarnopolsky & Berelowitz (1987) can be used for this purpose. Dahl (1985) has also reviewed the empirical studies in the diagnosis of the disorder.

Data on treatment is scanty. The final report of the Menninger Foundation's psychotherapy research project (Kernberg *et al*, 1972) is worth reading. With the expenditure of very considerable amounts of money and time, they were able to show that patients with borderline personalities were unsuitable for classic psychoanalysis, but that they could be treated with a modified, more structured form of psychoanalytic psychotherapy, which they called 'expressive psychotherapy'. The reader can decide whether or not this takes us significantly beyond what was said by Balint in 1968 or even by Stern in 1938.

There is no specific drug treatment for borderline personality disorder. Not surprisingly, several studies have shown that antidepressant and antipsychotic agents often provide symptomatic relief, but fail to deal with the underlying problem. Gunderson (1986) has reviewed this topic

There are several excellent criticisms of the borderline concept. Modestin (1980) provides a European perspective, and Akiskal *et al* (1985) an American one. Each paper argues that the so-called borderline patients could more profitably be subsumed under existing categories for personality and affective disorders.

The literature on childhood antecedents is scanty. The work by Mahler's group (Mahler *et al*, 1975) has methodological flaws, but is historically important and is much quoted by American psychoanalytic writers. The contribution of attachment theory (Bowlby, 1982, first published 1969) is potentially relevant, especially the work by Ainsworth's group (Ainsworth *et al*, 1978) on the Strange Situation

Finally, there are other, more relaxing ways of learning about borderline states. Characters in films such as *Five Easy Pieces* and *Scarface*, and plays such as David Mamet's *American Buffalo*, provide graphic illustrations of borderline psychopathology. However, the most sensitive and best account is in the novel *Tender is the Night*, by F. Scott Fitzgerald (1939) This book has it all: aetiology; course and prognosis; treatment difficulties, and implications for the doctor–patient relationship. It is said by some to be based on Fitzgerald's own marriage. If you have time to read only one reference about borderline states, read *Tender is the Night*.

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Books Reconsidered

The Psychoanalytical Theory of Neurosis: Otto Fenichel*

In the fly leaf, this book was described as a textbook of first rate quality and a standard reference for many years to come. However, psychoanalysis is not a static subject and, as such, does not lend itself to a definitive textbook with an inbuilt rigidity.

In a way, this book was already out of date when published in 1946. Following the controversial discussions in the British Psychoanalytical Society during the war, where different factions argued their cases, the Society formed three sub-groups in 1946: the followers of Anna Freud; the supporters of Melanie Klein; and an independent group who worked with such famous names as Balint and Winnicott. These new developments and their subsequent on-going scientific contributions would make the concept of creating a definitive textbook on psychoanalysis an impossibility.

The book can be looked upon as a résumé of the application of the early classic Freudian concepts to all spheres of psychiatry. The title is quite misleading as, in this book, Fenichel applies his analytic approach not just to neurosis, but to psychosis, psychosomatic disorders and personality disorders. He also comments on all varieties of treatment approaches fron counselling to ECT.

The book is not easy to read. When I started my psychiatric training some 20 years ago, having an interest in psychoanalysis, I bought Fenichel's book. Although I have used it in a reference sense over the years, at that time, I found myself unable to read it in a systematic way. The first consultant psychotherapist I worked for, strongly felt that it was not a suitable book for a beginner in the field of analytic psychotherapy due to its style.

Yet, the book carries a certain authoritative presence and has a classic, lasting quality to it. How is this to be explained? It seems that one needs to understand the man behind the book before turning to any more critical review of the contents. This information is to be found in the obituary to Fenichel given by Ernst Simmel in March, 1946, to a joint memorial meeting of the San Francisco Psychoanalytical Society and the Psychoanalytical Study Group of Los Angeles, as reported in the International Journal of Psychoanalysis, 1946.

Fenichel was described as the truest follower and best representative of Freud's teachings. To quote Simmel: "with Fenichel's death, Freud has died once more". Fenichel was described as not praising Freud, but quoting, explaining and defining him. At the time of writing the book, Fenichel had earned the reputation of *the* spokesman for the orthodox Freudian approach, with the facility to enumerate points in a cold and systematised way.

He was a widely read man who, even in his teens, had read some Freud, and although he had a medical training, his interest always lay in analysis. By the age of 21, he was reading his first paper as a guest speaker to the Viennese Society ("On a derivative of the incest conflict"). In 1922, he moved to Berlin to complete his analytic training and by 1933, when he left Berlin with the advent of the Nazis, he had already written 108 papers and published 34. In 1924, on joining the teaching staff in Berlin, although only 26, he had such a reputation as to be referred to as '*the* Dr Fenichel, already known in wide circles'. Fenichel described his own love for teaching as his 'teaching libido'. This love for didactic teaching can be seen to run through his book.

In 1933, he was invited by the Norwegian psychoanalysts to Oslo and in 1936, he went to Prague. Due to the threat of the Nazi invasion, he settled in California in 1938 and spent the remainder of his years teaching and training in San Francisco until his sudden death in 1946, shortly after the publication of *The Psycho-Analytical Theory of Neurosis*. By the end of his scientific career, he had given 200 papers, had had 72 publications, had written innumerable book reviews, but he is best remembered for the textbook under review here. The obsessional thoroughness of his approach to the subject can be appreciated by the size of the bibliography at the end of the 700-page book. It contains 1646 references, and yet he introduces them with an apology, saying a comprehensive bibliography had been prepared but that paper shortage necessitated limitations for the time being! The book was first published in 1934, but a second edition with a new introductory outline came out in 1946.

Fenichel would have seen his role as a teacher of Freud, clarifying the work of Freud to students. Also, in the early days, and ever since, there have been those who would try to dilute or refute Freud's insights. Thus, Fenichel viewed himself as the upholder of orthodox analytic theory against erosive tendencies. It is this view, held by Fenichel and others going to the USA with the Nazi persecution, that set the attitude of American post-war analysis, namely, to uphold classical analysis in a somewhat inflexible way, through the ego-psychology school of thought. It could be seen to uphold analysis against erosion but also to have a stultifying effect.

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*London: Routledge Kegan Paul (1966)

Freud's view was not one of a blind loyalty of theories. In his paper on narcissism (1915) Freud worried about not adopting too rigid an approach to metapsychology – this being the word used by Freud for viewing of the mind's structure in its most theoretical way. When talking of speculative theories, such as notions of the ego-libido, energy of the ego and instincts, Freud counsels for a "preparedness to replace by other notions as we apprehend things more clearly. For these ideas are not the foundation of science, upon which everything rests; that foundation is emotion alone. They are not the bottom but the top of the whole structure, and they can be replaced and discarded without damaging it".

As already stated, the book's title is misleading as it contains Fenichel's views of every branch of psychiatry. He even addresses the issue of statistics on therapeutic results of psychoanalysis. For non-analysts this might have been felt welcome, as here was an orthodox psychoanalyst of repute, prepared to give his views on every aspect of psychiatry, from an analytic position. However, his uncompromising style leads to structured over-simplification.

Despite his having extensive chapters on psychoses, perversions and character disorders, Fenichel insists his book only addresses neuroses. In the introduction he says "This book is *not* a textbook of psychoanalytical psychology, it limits itself to the theory of neurosis. It is true that neuroses, for the analyst, provide the most fruitful study in the realm of mental phenomena; after having studied the neuroses, it will be easier to study other mental phenomena. In this sense, this is perhaps a first volume of a textbook on psychoanalytical psychology".

At the outset, he categorically informs the reader of his authority. To quote again from the introduction, "We shall try to engage as little as possible in polemics, but concentrate, rather, on explaining that which already seems established. It is unavoidable that, in the choice of the material to be presented, in the decision as to which problems should be given more space and which less, and in the arrangement of the book, the personal beliefs of the author are reflected. However, since he hopes his scientific convictions are well founded, he is of the opinion that this will not be a disadvantage".

The reader is, to a certain extent, expected to be taught as an unquestioning pupil. Yet, it is Fenichel's, over and above Freud's and others', views that are expounded. For example, Fenichel repudiates Freud's concept of the death instinct. Fenichel writes: "There are many possible objections to his new theory. Here the following will suffice. The instinctual aim of destruction is the opposite of the sexual search for an object to be loved; of this there is no doubt. Questionable, however, is the nature of this antithesis. Are we dealing with basically different instinctual qualities, or is this contrast again a matter of differentiation of an originally common road? The latter seems more probable".

While Fenichel is by no means alone in his view, Freud's insight of a primary self-destructive instinct has enormous clinical application. Anyone working in general psychiatry and having to manage psychotic patients, are only too well aware of massive, self-destuctive forces in action. The concept of the death instinct was later elaborated by Klein, with envy taken as the external manifestation of the death instinct, and became one of the dividers of Kleinian and non-Kleinian theory. Bion later took the concept further, seeing the psychotic part of the mind dominated by the death instinct. He pointed out that it is at the point of being most in touch with emotional pain and needs that, in schizophrenia, the destructive forces are dangerously mobilised and can lead to a massive fragmentation of the mind and the process of thinking, in a way of which one, clinically, is so familiar.

Sometimes Fenichel's dogmatic style can be somewhat off-putting, leading to a need to hold on and digest statements further to get the substance from them. For example, talking about interpretations, he writes: "A valid interpretation brings about a dynamic change manifested in the subsequent associations of the patients and in his entire behaviour". One may argue that a narcissistic patient might ignore a valid interpretation, going on in an unchanged way. However, Fenichel also is making an important point, namely the need to look for the effects of an interpretation in the subsequent material that follows.

The more I read, the more I found myself wanting to argue against dogmatism. For example, he counsels against direct infant observation writing: "the temptation is great to apply concepts and ideas valid for higher stages of maturation to the behaviour of young children". Yet infant observation has become regarded as an important part of training for psychoanalytic and analytic psychotherapy courses, as well as child psychotherapy trainings. It enhances the capacity to observe non-verbal communication and report them, before giving consideration to their meaning. This helps towards learning the discipline of the neutral observer in analysis.

Perhaps he is most helpful in addressing the intricasies of Freud's metapsychology. With his Germanic background and thus his understanding of the original language, he conveys clear views on the meaning of instincts, drives, etc. However, the old-fashionedness of issues addressed is, at times, evident. In his chapter on mechanisms of defense he writes. "with regard to therapy, those types of actual neuroses that are due to an unfortunate sexual regimen do not need any other therapy". I don't think many analysts would think in this way nowadays.

Following a chapter on psychosomatic disorders, incorporating Alexander's well-known views of character-types underlying different disorders, he turns to the psychoses. In his chapter on manic-depressive states, there is a striking omission of two classical papers written in the 1930s by Melanie Klein, although he goes out of the way, at the back of the book, to say his references were up to 1943. These papers added crucially important concepts to our understanding of the dynamics of manic-depressive states. These included the concept of the manic defence, namely that manic states are states of denial of underlying unbearable depression and persecution, and the concept of manic reparation, in contrast to reparation proper. It would have been impossible for Fenichel not to have known these papers and their seminal importance. It is interesting that he quotes as many references to Melitta Schmideberg, as to her more eminent mother, illustrating the historical/political developments in analysis at the time, with the movement in America towards the ego psychology viewpoint and anti-Kleinian stance.

Paradoxically, Fenichel shows a refreshing stance towards the problem of tackling the inaccessibility of psychotic disorders. He stresses the need for pioneering work in the institutions. Freud, of course, had classified psychoses as narcissistic neuroses, namely that the libido was turned inwards, so that with the absence of transference, analytic interpretative work was not possible. Fenichel argues that in psychoses, "the analyst has no other means than to use the non-narcissistic remainder of the personality in attempts to increase patients object relationships sufficiently to start the analytic work".

Fenichel talked of a 'modification of analytic technique' necessary for this purpose, a view shared by other American analysts, such as Jacobson and Searles, involved with psychotics. In England, Rosenfeld was later to argue that there was a psychotic transference, concrete in form, which needed analysing by interpretation, and, in that sense, the basic analytic technique did not require modification in approaching psychotic patients

His summarised view of schizophrenia was to contrast it to neuroses: "Neuroses represent a regression to infantile sexuality; the psychoanalysis of neuroses brought about an understanding of infantile sexuality. Schizophrenia represents a regression to the primitive levels of the ego; psychoanalysis of schizophrenics will permit understanding of the evaluation of the ego". He emphasises Freud's concepts of regression, breakdown and reconstitution through delusional symptoms, to describe the evolution of the psychotic process.

The chapter on character disorders reflects the classic analytic thinking in the thirties, with the description of oral, anal, urethral, phallic/narcissistic and genital character traits. Again, Fenichel is refreshing in encouraging an approach to treatment of character disorders. He quoted Freud as saying that "a fairly reliable character" is one of the prerequisites of a successful analysis. Fenichel talks of firstly getting the patient to see that he uses his behaviour for defensive purposes in a repetitive way. If this can be achieved, then one might be able to make headway with some difficult character disorders.

His final chapters are on therapy and the prophylaxis of neurosis. He clearly differentiates counselling from psychoanalysis, but is not dismissive of counselling, emphasising the importance of the 'work of learning' and the 'work of adjustment'. He says that the patient "must acknowledge the new and less comfortable reality and fight tendencies to regression". In a style, as if directed for students revising for an examination, he has a section on contradictions to psychoanalysis, although many of those he mentions, such as age or schizoid personality, would nowadays be disputed.

It has been interesting to reconsider this book from the historical viewpoint. It shows that psychoanalysis has not stood still, but many advances in our knowledge on fundamental concepts and technique have taken place since Freud's death. The book may also illustrate how the attitude brought by Fenichel and others, fleeing from Nazi Europe, had a strong influence on the direction of development of post-war analysis in the United States. The uniqueness and thoroughness of the quality of Fenichel, as a teacher of psychoanalysis, does guarantee this book's place as a classic and a lasting reference source on early orthodox psychoanalysis.

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Book Reviews

Editor: Sidney Crown

Introduction to Psychodynamics: A New Synthesis. By MARDI J. HOROWITZ. London: Routledge. 1989. 259 pp. £25.00 (hb), £10.95 (pb).

Mardi Horowitz, an American psychiatrist and psychoanalyst, and well known for his earlier books *States of Mind* and *Stress Response Syndromes*, has now written an excellent introductory textbook on psychodynamics which brings together ideas from psychoanalysis, cognitive psychology, and biology.

The book is divided into two parts. The first part, which is longer, takes a developmental approach, showing how states of mind become organised into mental models, and how these then mould human relationships. In the second part, he applies these ideas to basic psychoanalytic concepts, such as the unconscious, mechanisms of defence, and psychic conflict.

Horowitz is at his best when describing states of mind, which he defines as a pattern of experiences and behaviours, which may have both verbal and non-verbal characteristics. He goes on to argue that these states become organised into a self-concept (i.e. one's conscious view of oneself) and a self-schema (i.e. the unconscious organisation of inner mental processes). These in turn lead to the development of particular role-relationship models, which result in particular ways of relating. When restricted or recurrently maladaptive ways of relating develop, problems will arise.

These ideas are illustrated with excellent clinical examples, which are easy to follow, as well as a number of diagrams, with which this reviewer had some difficulty.

In the second part of the book he takes us on a quick tour of the conscious and the unconscious, parapraxes, and a long list of defence mechanisms. Again, these are illustrated with case studies and diagrams, punctuated by some sharp comments and questions (e.g. Why is consciousness not under full conscious control?).

The book is not always easy to follow, partly because Horowitz avoids some comfortingly familiar jargon, and partly because he draws from so many different schools of thought. At other times his ideas seem deceptively and seductively simple. Overall I would strongly recommend the book, because Horowitz's contribution to the understanding of internal and interpersonal relationships is original and stimulating. He not only presents some old ideas in new and refreshing ways, but also introduces some very interesting new ideas of his own on the workings of the mind, and the clinical implications thereof. Furthermore he attempts (with some success) to integrate ideas from disciplines whose adherents are all too often in conflict with one another. His work should therefore be of interest to psychotherapists from a number of different schools, as well as to psychiatrists, child psychiatrists and psychologists.

MARK BERELOWITZ, *Consultant Child Psychiatrist, Camberwell Child Guidance Clinic and the Maudsley Hospital*

The Technique and Practice of Listening in Intensive Psychotherapy. By RICHARD D. CHESSICK. New Jersey: Jason Aronson Inc. 1989. 296 pp. $27.50.

Just as the choices for mathematical computation have increased from counting on fingers to the use of complex computers, so over the past century, has the range of psychoanalytic theory and technical options also expanded.

In his latest book, Chessick explores the technique and practice of listening in psychotherapy in his usual novel and succinct style. Freud (1912) suggested that in psychoanalysis the listener turned his own unconscious into a telephone receiver adjusted to the transmitting unconscious of the patient. Chessick proposes that the therapist should metaphorically view his receptive state as rather like a television set with listening being constantly scanned along five different channels representing five main theoretical approaches. These channels are: (a) Freud's drive/conflict/defence orientation; (b) the object relations approach of Klein and Bion in particular; (c) the phenomenological or sociocultural approach exemplified by Fromm, Sartre, Lacan and Foucault; (d) self psychology expounded by Kohut, and the works of Fairbairn, Winnicott, Gedo and Laing focusing on empathic listening; (e) the interactional approach of Sullivan and Gill with its emphasis on 'here and now' factors in the treatment setting.

Being prepared to switch listening channels, Chessick believes, requires a preparedness to move away from a personally-tuned rigid 'one channel' theoretical stance, and increases the listener's ability to hear and understand more clearly the transmissions from the patient's unconscious.

Chessick explores this five channel approach in chapters on listening to different categories of patients–psychotic, borderline, neurotic, narcissistic and 'difficult'. Surprisingly for an author who has written very widely on this topic, I found myself disappointed by his section on listening to the borderline patient. However, I was impressed by his views on listening to the 'difficult' patient.

The final chapter "Learning to listen' seemed to highlight and crystallise some of Chessick's ideas from more than thirty-five years of clinical practice, and reading these I hoped that they could be expanded in a further book.

This is a richly crafted, clearly written book which will appeal to and inform psychotherapists at all levels of training and experience.

JOHN BIRD, *Consultant Psychiatrist,*
Cheadle Royal Hospital, Cheadle, Cheshire

Issues in Psychobiology. By CHARLES R. LEGG. London & New York: Routledge. 1989. 224 pp. £30 (hb), £12.95 (pb).

Any book on a sober scientific topic which opens with a reference to the *Hitchhiker's Guide to the Galaxy* deserves our serious attention. The reference concerns the need to ask the right questions if, seeking the answer to 'life, the universe and everything', we hope for a more enlightening answer than '42'. This book, we are told, will focus on questions rather than answers, but we should not perhaps take this too literally for the book contains a wealth of carefully detailed information.

Psychobiology as addressed by this book concerns the relationship between cerebral functioning and human behaviour and experience. Two chapters set the scene. The first one is on the use of animal models, and both here and throughout the book, the author stresses their value and limitations in elucidating human problems. The second one concerns methodology, and the conclusion that, while different approaches have their drawbacks, findings emerging from all types of studies have enhanced validity.

The main body of the book covers perception, motivation, emotion, memory, plasticity and consciousness. In several of these subjects, the author focuses on one well-researched area. For example, the discussion on perception concentrates on visual perception, the other sensory systems being seen off in four paragraphs. Similarly, motivation is confined to that which is biologically oriented, although the consideration of blood glucose levels and cellular dehydration is a far cry from the more usual reinforcement concerns of the practising behaviourist (such as the present reviewer). Nevertheless, it is fascinating to read of studies showing that the triggers of eating behaviour may be quite different in the starving and the merely hungry; and that salivation not only aids digestion but makes us hungrier so certain foods will act as reinforcers even where there is no deprivation. Indeed, here the practising behaviourist gets a glimpse of land on the horizon. A water-deprived animal will stop drinking long before blood plasma changes take place and precisely when the amount taken is that required for homeostasis. The beauty of it makes one feel quite faint!

And so to emotion (=fear), memory, and the plasticity of the brain – true recovery is uncommon but much can be achieved given time and, more particularly, training – and consciousness. Knowledge of these topics may be transformed in the next decades but the fundamental issues may remain those described here.

The book is clearly and effortlessly written, entertainingly even at times. In a work of this weight, this is a distinct plus. Nobody would pretend it is an easy read but it repays reading. As a ignoramus in the field I feel I have caught up a little, and I recommend it.

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Reliability and Validity in Neuropsychological Assessment. By MICHAEL D. FRANZEN. New York: Plenum Press. 1989. 304 pp. $35.00.

In this book, Michael Franzen addresses the issues of reliability and validity in relation to a wide range of neuropsychological assessment instruments. He is concerned with identifying areas where research studies are required, the results of which will enable neuropsychologists to make better-informed choices between different measures.

Chapters 1–5 provide a detailed discussion of theoretical and practical issues associated with the concepts of reliability and validity. It is stressed that these concepts are of paramount importance to contemporary clinical neuropsychologists who are asked to provide much more sophisticated assessments than their predecessors.

Chapters 6–20 focus on specific tests or types of tests. In each chapter, test details are described, together with a meticulous review of the available reliability and validity studies and a useful conclusion. Where studies are lacking, Franzen highlights this. I found coverage of the Wechsler's Adult Intelligence Score (WAIS–R). Wechsler Memory Scale, and Halstead-Reitan Neuropsychological Battery of particular interest.

There is no doubt that this is an extremely competent, timely review. However, at the risk of nit-picking, I would have liked more tables summarising statistical information, as on occasion I felt swamped by figures. Also, the book ends rather abruptly, and would have benefited from a final chapter providing a general summary and overview of the quality of available test instruments.

A more significant criticism concerns the limitations of test coverage from the point-of-view of a British readership. Franzen discusses many tests which are not readily available in the UK. He also includes chapters on the Minnesota Multiphasic Personality Inventory (MMPI) and Rorschach which, although of interest, are rarely used by British neuropsychologists. This, together with the omission of a number of important British tests, will undoubtedly limit the popularity of this book outside the USA.

Nevertheless, Franzen is to be commended for tackling an important topic which is fundamental to meaningful assessment. It is sobering to realise the paucity of good studies on so many well known and well used tests, and this underlines the importance of this review. There is much of value here both to students and to experienced neuropsychologists.

Richard M. Blunden, *Senior Clinical Psychologist, Department of Psychology, The London Hospital*

Love is Never Enough. By Aaron T. Beck. London: Penguin. 1989. 323 pp. £4.99.

This is a self-help guide, with which "couples can overcome misunderstandings, resolve conflicts and solve relationship problems through cognitive therapy". The professional reader, therefore, will find no direct advice about managing consultations, nor data concerning the effectiveness of Beck's methods. He or she can still profit however from the analysis of the roots of distorted communication that occupy the first half of the book, and the range of procedures for assessment of difficulties and their repair that are outlined in the remainder.

The book stands independently of Beck's other work. It argues that forms of symbolic distortion familiar to cognitive therapists can be singularly damaging once they taint communications between intimate partners, with unnecessary assumptions of incompatibility resulting. Beck's prescription for a partnership's staged recovery depends upon rebuilding its forgotten strengths, allied to an active re-examination of the fears and assumptions that led to each partner's behaviour being evaluated so negatively. A range of therapeutic possibilities are described, some appropriate to quite entrenched degrees of marital pathology. Beck is careful to supply hope and strategic advice also to the embattled reader who, at least initially, lacks co-operation from their partner.

This is a welcome book that develops its themes by the use of simple vignettes. Like the title, the book's style is clear if prosaic, making its length tell at times. Its cognitive perspective naturally benefits some analyses more than others: it copes well with anger (chapters 7 and 9), seems rather weaker on attraction (chapter 2), while readers are likely to have to go elsewhere for sex (chapter 18). Practically speaking, Beck supplies a very flexible set of therapeutic tools (and a measure of healthy disdain for the trite formulae of other self-help writers), prompting the reviewer to wonder how well the best-intentioned of couples would cope without some further guidance in their use.

Indeed, clinicians should consider recommending the book to suitable patients in order to enhance the usefulness of consultations, rather than as a complete substitute for them. The pragmatic insights it contains deserve a wide readership among non-psychiatrists with counselling or psychotherapeutic responsibilities. The volume represents excellent value, and deserves to remain in print for some time.

Chris Mace, *Senior Registrar in Psychiatry, St George's Hospital, London*

Philosophy of Psychology. By Daniel N. Robinson. New York: Columbia University Press. 1989. 188 pp. $14.50.

This book is aimed at prompting those who are involved in the study of human behaviour to do so with a greater sensitivity to philosophical inquiry of the concepts we use and the way in which we use them. It contains five chapters. The first, entitled "The armchair and the laboratory", addresses the importance which philosophical modes of questioning can play in helping to shape a clearer way of thinking about the concepts we use. The second, entitled "Determinism, hard and soft", deals with the subject of causation against the background of the views of some of the earlier and more recent notable philosophers. Chapter three, "Reductionism: models, metaphors and similes", debates the issue of 'private languages' in relation to the somewhat more familiar topics of behaviourism, mentalism and artificial intelligence. Chapter four, entitled "Explanations", covers the wider arguments of how we explain the phenomena of human actions, and debates some of the different perspectives, including psychoanalytic, mechanistic and teleological. The fifth and final chapter is on "Ethics and psychological inquiry" and brings us up to date with some of the more recent ethical issues (e.g. 'gay rights', 'animal rights', 'free enquiry').

The style of writing makes for fairly easy reading. Although there is nothing new in the book it nevertheless does provide a coherent argument for trying to encourage the reader to think more deeply about the concepts used in human science. In the author's words, "As with statistics and logic, the speciality of philosophical psychology is likely to be a tool for the many; as a set of guidelines for clarifying certain concepts, for assessing certain strategies, for weighing the merits of certain arguments" (p. 8). The book is more likely to appeal to the serious thinker than the casual reader.

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Treating Schizophrenia. By WERNER A. MENDEL. London: Sage Publications. 1989. 244 pp. £24.95.

A Clinical Guide for the Treatment of Schizophrenia. Edited by ALAN S. BELLACK. New York: Plenum Publishing. 1989. 329 pp. $39.50.

These two books not only emanate from across the Atlantic but also from authors whose practices of psychiatry appear somewhat different. They will thus probably appeal to different audiences. Although both profess an eclectic view of treatment, Mendel's book seems to approach this very much from the view of office-based psychotherapy and Bellack's one from that of an active social psychiatry model. Neither model really equates very well with current psychiatric resources in the UK, particularly in inner-city areas where the attempt to keep track of patients and to keep them on maintenance medication can be difficult enough with scarce resources. However, there is some merit in these books, particularly that of Bellack.

Mendel's book makes a promising start, stating that it is written from the long-term observation of 497 patients. Other than some general description of these, this fund of information is not then elaborated upon or made great use of. What follows is a rather personalised view of treatment interspersed with occasional homily and sadly little scientific observation. There is no doubt that much of what is said is true and some points are worth being reminded of. However, the book is rather lengthy for the content and concentrates too much on a few individual cases. A large section of 41 pages is taken up by a description of a case (Ellen West) originally recorded by Ludwig Binswanger. One of the more interesting points in this is that the patient saw both Kraepelin and Bleuler who disagreed on the diagnosis.

The chapters of Bellack's book represent the views of different experts writing on their own areas of expertise. They are fairly up-to-date as far as the current literature goes and read fairly coherently together. Following an opening overview, the next two chapters give a fairly concise review of pharmacological management, the one by Kane providing and up-to-date summary of recent thinking and developments. The subsequent nine chapters each focus on different types of social or behavioural intervention and include aspects of family therapy, crisis intervention, case management and varying approaches to the problems of accommodation and admission to hospital. The one area notably absent from all of this was work therapy. The idea of providing structured activity through workshops or day centres, particularly for more disabled patients, is barely mentioned.

Furthermore, one is left with a strong feeling that while many interesting approaches are discussed, for example in the chapter "Community residential treatment", these often have a high staff·patient ratio and somewhat selective entrance criteria. Therefore they may not be readily transferable elsewhere.

The former of these two books is for those who like to read of another's fairly personalised experiences in treating patients and is not useful for trainees. The latter book gives an overview of a variety of approaches within the social psychiatry sphere and thus may be of use to those wanting more than textbook detail of these

STEVEN J. COOPER, *Senior Lecturer, Department of Mental Health, The Queen's University of Belfast*

Schizophrenia: Scientific Progress. By S. G. SCHULZ and CAROL A. TAMMINGA. Oxford: Oxford University Press. 1989. 434 pp. £50.00.

This is a complicated book, possibly due to uneven editorial control. The authors are nearly all from the USA (18 of 122 are from Europe), and only 60% seem to be medical. Perhaps, in contrast to the European situation, this reflects a lack of US medical interest in psychosis until recently. Not surprisingly, in view of the North American influence, DSM–III dominates throughout.

So many research topics are covered, that it is possible to mention only a few. The proposal to replace diagnosis with domains or clusters of co-varying symptoms is rather at variance with the suggested classification of mental disorders which leans heavily on phenomenology and aetiology. The significance of positive and negative symptoms, and their relationships to brain ventricular size, prognosis and response to therapy are discussed (although the attempted subclassification of negative symptoms is unconvincing).

One of the book's best features is the section on genetic and family studies. Clear evidence is presented that genetically, schizophrenia and affective illness are distinct but, although familial is distinguished from genetic, little support is adduced for the current vogue for unitary psychosis. Also of interest is the excess of winter- over summer-born schizophrenics in Scotland, while the reverse is true of affective illnesses. Further light is shed on the aetiology of schizophrenia by a South London study which shows that those with evidence of dilated brain ventricles and a birth history of obstetric complications have a smaller genetic loading than patients free of these stigmata.

There are excellent sections on those rather arcane topics of computerised axial tomography (CT scanning), magnetic resonance imagery (MRI) and positron emission tomography (PET scanning). Not so convincing were the chapters dealing with the animal and other models of schizophrenia. I have always doubted the strict relevance of 'models' of schizophrenia, and since animals cannot talk, I fear we may be maligning them by attributing disorders of thought and emotion to them

Indeed, at the heart of this book there are fallacies: we do not know whether schizophrenia is one, two or more conditions (Bleuler talked of the schizophrenias); the use of models, especially animal models, may be downright misleading; and our clinical insights are based on the acute observations of Kraepelin, Bleuler, Schneider and others rather than animal and laboratory evidence. In the absence of a generally agreed pathophysiology for schizophrenia, is it not premature to rush to judgement in the manner of some sections of the book?

A valuable observation is that contrary to widely held beliefs, dopamine receptor blockade is not essential for the development of tardive dyskinesia (TD). In fact, in TD, several dopamine receptors may remain unoccupied and a gamma-aminobutyric-acid (GABA)-mediated effect is postulated. TD may also be seen in bipolar patients and is linked with spontaneous movements, cognitive impairment, affective blunting, enlarged ventricles, negative symptoms and increasing age: but not, surprisingly, with chronicity of illness. Clonidine is recommended as a neuroleptic and as treatment for akathisia.

This book is not cheap and thus may be used as a reference volume rather than as a textbook.

M. W. P. Carney, *Consultant Psychiatrist, Northwick Park Hospital, Harrow*

The Treatment of Psychiatric Disorders. By William H. Reid (with contributions from George U. Balis, James F. Wincoff and Jerry J. Tomasovic). New York: Brunner/Mazel Publishers. 1989. 432 pp. $35.00.

This is an expanded and updated revision of a previous textbook, *Treatment of the DSM-III Psychiatric Disorders,* and is keyed to the DSM-III-R diagnostic system. It is described as suitable for 'psychiatric and non-psychiatric physicians, residents and medical students, as well as non-medical mental health professionals", but it seems to be written specifically with the psychiatrist in mind. Its four sections deal with childhood and adolescence, organic mental disorders, psychoactive substance use and (most extensively) other adult disorders.

The format inevitably poses problems. It assumes a familiarity with the DSM-III-R classificatory system and a good knowledge of the epidemiology, aetiology and clinical features of the disorders for which treatments are described. The author warns the reader against becoming enamoured with the apparently 'cookbook' style of this presentation, but the temptation is there to pursue the naive assumption that for each psychiatric diagnosis there is a specific recipe. Since every disorder is accorded at least brief consideration, there is much repetition, and overall probably inadequate encouragement to the reader to think about general principles of treatment. In addition, the perspective is that of a physician who has yet to develop an interest in multi-disciplinary management and the imaginative use of community resources.

Notwithstanding the limitations, this text offers clarity of expression, sensible treatment suggestions, good references (not all of them to North American literature), and a particularly good account of delirium. Factual information is almost invariably accurate, but *limbitrol* (p. 174) is wrongly designated a combination of antipsychotic and anxiolytic, while temazepam and triazolam (p. 239) are questionably welcomed as increasing the rapid eye movement (REM) content of sleep. Furthermore, drug dosage regimes are sometimes quoted in archaic abbreviations.

Of particular interest are the occasional differences which emerge in treatment practices on opposite sides of the Atlantic; depot medication for schizophrenia receives no support; short-acting barbiturates are advocated for brief reactive psychoses; for anxiety states, benzodiazepines are the anxiolytics of choice, and cognitive therapy is given curiously scant attention; and lithium is to be discontinued in most patients undergoing electroconvulsive therapy (ECT).

Such thought-provoking differences give spice to this otherwise uncontroversial book. Worth buying for a library? That would be difficult to justify.

David Julier, *Consultant Psychiatrist, Littlemore Hospital, Oxford*

Current Reviews in Psychiatry: Adolescent Psychiatry. By Peter Hill. London: Churchill Livingstone. 1989. 346 pp. £29.95.

This is a clear academic work in short sections. It was easy to find my way around. Lists and tables are used well, for example, in clarifying the powers of courts. Crucial issues, such as how to bring the fatal riskiness of overdosing into the open, are also presented well. Although when Hill describes the power of the nurses' hug, the taboo on therapeutic touch is broken, I was disappointed that touch had no entry in the index. There are nice tips about interviewing adolescents.

Compared with two good predecessors (John Evans' *Adolescent and Pre-Adolescent Psychiatry* (1982) and Derek Steinberg's *The Clinical Psychiatry of Adolescence* (1983)), this book is cheaper, smaller, and emphasises diagnosis rather than treatment. Tourette syndrome is not mentioned by Evans, is given two-and-a-half pages by Steinberg, but a whole chapter with 26 references here. The chapter on suicidal behaviour is a particularly good example of the bringing together of epidemiology and mental state phenomena; it includes an excellent and non-inflammatory section on something that always distresses me, namely cutting. Because Evans' and Steinberg's works were good too, I put on my most critical hat. Inevitably, I did find some things to be disappointed about: just a mention of AIDS, but nothing in the index; no mention of Russell's evaluation of family therapy in anorexia or Shaffer's work on the influence of TV on suicide; and no sufficient discussion on cognitive and behaviour therapy.

Peter Hill is one of the liveliest and most original presenters of child and adolescent psychiatry. Unfortunately the dry academic style imposed by *Current Reviews in Psychiatry* sometimes submerges him. He becomes "(Hill)" or "Hill (1985)" most of the time, but does gloriously manage to burst out as a person with statements beginning with the pronoun 'I' and with opinions about many things. He does give his own views clearly, generally makes good sense, and quotes widely. Occasionally my understanding of cited works is different from his, as with Freud on sexual abuse, but this was not a problem which got in the way of my enjoyment of this book, nor for that of the colleague who asked to borrow it. Peter Hill has given us a good and enjoyable book.

PETER BRUGGEN, *Consultant Psychiatrist, Hill End Adolescent Unit, St Albans*

Experiential Therapies for Eating Disorders. Edited by LYNNE M. HORNYAK and ELLEN K. BAKER. New York: Guilford Press. 1989. 339 pp. $35.00.

This is a collection of papers by therapists from a range of disciplines on their approaches to the treatment of anorexia and bulimia. They are brought together under the heading of 'experiential' therapy, defined by the editors as "treatment techniques, based on psychological principles, that are developed and used with the specific intention of increasing clients present awareness of feeling, perceptions, cognitions and sensations; that is, their in-the-moment experience". One could quibble about whether the principles behind some of the therapies are actually 'psychological' in the sense of a scientific approach, but a more fundamental concern about the definition is that it concentrates on the technical aspect, how to evoke feelings, rather than on how to resolve them. Although the editors emphasise the need for both phases, and some contributors offer useful suggestions, many papers reinforce the sense of reading about exploratory techniques rather than comprehensive therapies. The coverage of the book is, however, broad, ranging from hypnosis to art therapy, from exposure with response prevention to psychodrama, and from dance and music therapy to family sculpting. Overall, there is some bias towards therapies rooted in the creative arts.

The authors acknowledge that there is little systematic research or empirical evidence for the effectiveness of many of the techniques presented, and indeed most of the contributors are therapists rather than researchers, giving accounts of clinical cases and personal styles of work. All are from the USA, many working in private practice and presumably with a self-selected group of clients who see a particular therapy modality as having relevance to them. It seems doubtful whether some of these experiential therapies could be applied routinely to a less selected population.

In summary, this book offers a glimpse into worlds of therapies which may be distant from one's own. Most readers will feel sceptical of at least some of these, but could also find new ideas about ways to bring problematic feelings and patterns of thought into the 'here and now' of their own therapy sessions. Apart from specialists in creative/artistic therapies, however, few will wish to do more than dip into this volume selectively.

KENNETH GORDON, *Head of Psychology Services (Mental Health), Winchester Health Authority, Winchester*

Innovative Trends in Psychogeriatrics. Edited by J WERTHEIMER, P. BAUMANN, M. GAILLARD and P. SCHWED. Basel: Karger. 1989. xii + 120 pp. £52.30.

That old-age psychiatry deserves specialty status can be demonstrated by the breadth and scope of this book. In it are collated papers presented at an International Symposium of Psychogeriatry (Lausanne, 1988) which focused on interdisciplinary contact and exchanges in the gerontological and geriatric domains.

Under four general themes, 16 papers are presented, reflecting the growing concern of researchers, teachers and planners with the psychiatric problems presenting in old age. Experts from a wide range of backgrounds contributed. In the first section, "Dementia and built environment", a geographer, architect, an environmental and organisational psychologist and a geriatrician individually examine the impact of urban and institution environment on the demented elderly. The need to integrate rather than isolate homes for the elderly within the community is highlighted; all too often places of work are of social and cultural activities are placed at an inaccessible distance with a resulting loss of stimulation and interaction.

Lead by Arie, the who, what and when of teaching old-age psychiatry in Britain, Germany, France and Canada are discussed. A multifaceted opportunistic approach is emphasised ranging from 'instant aging' in which students 'experience' one of the common impairments of extreme old age, to full teaching programmes addressed to primary health care workers.

As our knowledge of the dementias grows so too does the need to refine their definition and classification. In a well written section which should appeal to all psychiatrists, Flament-Durand gives the neuropathological point of view, while that of the clinician is provided by Blessed. Sartorius looks at the classification of the dementias in the ICD-10 proposals while also providing a first hand account of dementia related research co-ordinated by the World Health Organization.

The final section is devoted to recent advances in the psychiatry of old age, in particular in the psychodynamic, pharmacological and neuroimaging spheres. The use of neuroleptics and antidepressants in this age group is reviewed while the need to integrate this approach with the psychotherapeutic and psychosocial is stressed. In a lucid account of functional imaging in dementia, the clinician will be disappointed to read that these techniques, while expanding our knowledge of the underlying pathophysiology, are not yet useful as diagnostic tools.

It is inevitable in a work consisting of 16 short papers and covering such a wide canvas that some parts appear overly brief. Moreover, because of diverse authorship the papers are presented with varying complexity, some of which demand the reader to have more than a basic knowledge of the subject.

In a field where so much of the work is community based, it is surprising to see little or no attention given to the many recent developments in this area. Apart from this significant omission it deserves recommendation for the balance it has achieved and should appeal to all those interested in the development of psychiatry of old age.

CON KELLY, *Senior Registrar in Old Age Psychiatry, St Charles Hospital, London*

Empathic Attunement: the "Technique" of Psychoanalytic Self Psychology. By CRAYTON E ROWE and DAVID MAC ISAAC. New Jersey: Jason Aronson. 1989. 338 pp. $30.00.

This book, written by a social worker and a psychologist, both founding members of the New York Institute for Psychoanalytic Self Psychology, is a kind of 'Kohut without tears'. Kohut, a loyal supporter of Freud until the late sixties, suffered, with the publication of his first book *The Analysis of the Self* (1971), rejection by the psychoanalytic establishment. By his death ten years later, the importance of his contribution both to theory and practice was becoming acknowledged. Throughout the eighties his influence has grown. In writing this book, the authors have taken a further step towards bringing Kohut's ideas to a wider public.

The first third of the book is devoted to a simple and concise exposition of Kohut's major principles. The remainder is taken up by the description of two courses of therapy (we are not told by whom), lasting six and eight years respectively, of patients who suffered from narcissistic personality and borderline personality. Kohutian therapy strikes me as a very indulgent kind of therapy, aimed at providing, within the self-object transference, the mirroring which the patient lacked during the appropriate phase of infancy, and probably later as well. The mirroring in adult therapy however is provided not so much by the 'gleam in mother's eye' as by the therapist's correct perception of what the patient is trying to say. Being correctly understood is a potent component of therapy, which is achieved through the all important process of empathic attunement. When, on the other hand, an idealising transference develops the therapist must allow him/herself to be adored.

The distinction which Kohut draws between the "experience-near" and the "experience-distant" approach to patients is of immense importance not just to psychotherapy but to all caring involvements, and also to research. To be "experience-near" to the patient is to want to be close enough to him/her to feel what he/she feels. To be "experience-distant" is to keep him/her at bay by a kind of diagnostic interrogation. In the former you relate on his/her terms, in the latter you compel him/her to relate on yours.

JOHN BIRTCHNELL, *Scientific Officer and Senior Lecturer, MRC Social and Community Psychiatry Unit, Institute of Psychiatry, London*

Developmental Breakdown and Psychoanalytic Treatment in Adolescence: Clinical Studies. Edited by MOSES LAUFER and M. EGLE LAUFER. London: Yale University Press. 1989. 217 pp. £19.95/$34.

This book represents part of a study being carried out at the Brent consultation centre. Its core is formed by nine chapters, each detailing the breakdown and subsequent treatment of extremely disturbed and self-destructive adolescents. Their self-destructive behaviour is largely seen as an attack on their sexually mature body which puts unbearable demands on the fragile equilibrium they might have achieved by the end of the oedipal phase. It is conceived that much of the otherwise incomprehensible presenting material in such patients may be understood as attacks on the sexually or at least genitally mature self which take the form of suicide, mutilation or psychosis. Such behaviours, when they cannot be understood in terms of their purpose in re-establishing pre-genital forms of relating, are treated as incomprehensible by those from whom help is sought, in such a way as to increase alienation, and are often treated by inappropriate means which have the effect of reinforcing the attacks on the emergent adult self and enhancing the wished-for but feared infantile helplessness.

The nine case studies all make exemplary reading for general psychiatrists and illustrate the value of surviving the constant attempts of extremely disordered patients to enlist their despair and to give way to treatments that confirm the idea of personal incoherence and fragmentation. This is true whatever the 'diagnosis', however its aetiology might be understood, and even though in the case of the nine patients described, most would be described as severe borderline personalities. The studies also underline the importance for general psychiatrists of a thorough grounding in psychodynamics, of the importance of adequate supervisory support and of adequate attention to the patients' network. These are as true for adult patients as for the adolescent patients described in this book and the political implications are obvious in a climate which threatens all three.

I was not sure, however, from an analytic viewpoint, whether the study was so successful in establishing a particular meaning for psychopathology in adolescence (as opposed to childhood or adulthood) or that the borderline features described in these nine patients were any more or less amenable to intensive treatment than comparable patients in adulthood. Similarly, such patients at any stage of life make demands in the transference/countertransference interaction which many will not wish to meet and it was difficult to judge from the material in this book whether the patients described made demands of greater intensity as adolescents than would other groups.

Whatever the case, however, the book stands as a testament to the courage, tenacity and talent of the clinicians who tolerated these encounters with their patients and in so doing mitigated their pathology to a significant extent.

R. R. N. CARVALHO, *Consultant Psychotherapist, St Mary's Hospital, London*

Therapy for Adults Molested as Children: Beyond Survival. By JOHN BRIERE. New York. Springer Publishing. 219 pp. $29.95.

This is an interesting book, the main focus of which is in addressing therapeutic techniques and practical and clinical problems that arise in treating adult survivors of childhood abuse. It is mainly directed at the therapist.

The book consists of ten chapters. The first two chapters provide a concise and detailed review of evidence that sexual abuse leads to significant psychiatric pathology in later years and discusses the specific problems that are likely to occur. Briere then outlines the basic philosophy used in treating survivors of sexual abuse which includes advice such as having respect for the patient and setting clear limits and boundaries in therapy (e.g. not inviting the patients home for tea, or letting them babysit for the children), advice which is appropriate for all patients, whatever the diagnosis.

Briere spends some time on the sensitive area of possible patient abuse within therapy. Because of the client's tendency to sexualise all close interactions, he believes this may lead to inappropriate acting out by male therapists and believes that work with female survivors should mainly be tackled by women However, there are risks with female therapists of overidentification with the client resulting in the therapist becoming anxious and overinvolved or alternatively detached and critical

He recommends specific therapy techniques which, although presented in irritating jargon (presumably because the book is aimed at an American readership), are probably quite sensible when translated into comprehensible English (e.g 'disrupting the abuse dichotomy', 'therapeutic restimulation' and 'playing the observer game with the client') He also looks at various obstacles within the client and therapist that may block therapeutic progress. transference and countertransference issues, various forms of dissociative phenomena, pseudoparticipation, and the client's tendency to self-destructive behaviour

The last chapters look at the alternatives to individual therapy, group and family therapy and discuss the benefits and the risk that both might entail Finally, he addresses the problem of how therapists can protect themselves from becoming too disheartened in the process of treating sexual abuse and recommends remedies that I would wholeheartedly support, such as taking frequent holidays and avoiding a caseload that consists solely of victims which, he warns, sooner or later grind the therapist down In the appendices he includes some information on psychological testing of sexual abuse survivors including the Maudsley Multiphasic Personality Inventory (MMPI), the Roschach and the trauma symptom checklist, although these have little relevance to the rest of the book which is essentially not empirical or research orientated in nature.

Although some of the text appears somewhat pedantic in tone and repetitive in its arguments, I would recommend this book to any therapist who find themselves at a loss when knowing how to treat the long-term effects of sexual abuse (who doesn't?). The underlying message is clearly set out, sensible but not unduly optimistic There are many interesting ideas and issues raised, particularly related to the dangers of recreating the abuse in the therapeutic relationship Briere only addresses minor psychiatric disorder however, and not possible links between early sexual abuse and the later development of major psychiatric pathology or the possible need for chemotherapy. Furthermore, while recognising that men can be abused as children, the book is very definitely aimed at the problem of the female survivor. Finally, there is an underlying assumption that the free expression of an abusive experience for the survivor is inevitably going to be a cathartic and essentially healing process. I am not certain that the case for this has been proved beyond doubt or whether, in certain cases any information disclosed needs to be reburied without an ensuing post-mortem.

GILLIAN MEZEY, *Lecturer in Forensic Psychiatry, The Bethlem and the Maudsley Hospital, London*

Child Maltreatment: Theory and Research on the Causes and Consequences of Child Abuse and Neglect. By Dante Cicchetti and Vicki Carlson. Cambridge: Cambridge University Press. 1989. 814 pp. £40.00 (hb)/£15.00 (pb).

The editors of this book describe in their preface how professional perceptions of child abuse have changed rapidly. Widespread recognition of the nature, high prevalence and consequences of abuse took place only from the 1960s onwards. Following this, a variety of models designed to explain the phenomena appeared. The first were narrow in their focus (which could be seen in the use of the term 'battered baby), concentrating entirely on parental pathology. During the 70s a wider perspective was taken and the importance of social factors was acknowledged. Cicchetti and Carlson, however, feel that the most important advances in our understanding have been much more recent and involve the use of ecological and transactional models to study child abuse and maltreatment. The reason for their enthusiasm is that these new approaches bring the study of abuse, both its causes and consequences, into the wider fields of child development and developmental psychopathology, where there has been major progress. The intention behind this book is to bring together the major theoretical and practical advances of the past ten years.

At first glance this 800-page densely-referenced work looks very off-putting. In fact, however, the writing is of a quality which allows the reader to absorb relatively painlessly a vast amount of complex information. Each of the 23 chapters is written by an acknowledged expert in the field. The standard of scholarship is uniformly high. There are three sections. The first deals with history and definitions. The chapter by Kaufman & Ziegler examines the intergenerational transmission of abuse and neglect and goes on to demonstrate that the widely held view that the abused child automatically becomes the abusing parent is a gross oversimplification of a complex set of relationships. The second section deals with parental and contextual influences on maltreatment. Here, a great deal of interesting work on the determinants of parenting, both adequate and abusive is presented. The third section, which takes up almost half of the book, brings together in an authoritative way a great deal of work on the developmental consequences of child maltreatment, an area often hardly touched. The topics include effects on peer relationships, on qualities of attachment and on the social cognitions of the abused child. There is an interesting account by Cicchetti on how research on abuse has influenced the more general study of child development.

This is not a treatise on clinical practice but a thought-provoking book showing how research should be influencing and possibly changing our approaches to this complex and disturbing topic. It should be available in any centre where professionals are trying to come to terms with child abuse. Highly recommended.

Stephen Wolkind, *Consultant Child Psychiatrist, The Maudsley Hospital*

Research Methods in Psychiatry – A Beginner's Guide Edited by Chris Freeman and Peter Tyrer. London: Gaskell. 1989. 248 pp. £10.00.

It is seldom that a book is exactly what is needed. However, in this case it is an enormous pleasure to see the College publishing a book of such value. There can be little doubt that trainees engaging in research for the first time will find it immensely useful, as will many of their senior colleagues, who could use it both to catch up on a research training they may never have had, and also as a reference for particular techniques such as power calculation.

The book arises naturally from the course on research methodology which Chris Freeman and Peter Tyrer have been running on behalf of the College. This has obviously been very successful and there is no doubt this book will be a useful addition to the reading list of that course. Hopefully, it will not replace the course for too many trainees. Research is the sort of activity which requires regular discussion with colleagues. Very few people can manage to do effective research while interacting only with a book.

The book is set out in a number of obvious, although not overstated, sections. There is a good chapter on getting started in research by the editors which takes us right back to square one. This is followed sensibly by a chapter on the methodology of clinical trials in psychiatry which, apart from full-length books such as Pocock's, is one of the best accounts I have seen. It includes a brief statistical discussion of power and confidence intervals. The next chapter deals with research methods appropriate to biological psychiatry, which includes a brief run-through of a number of rather disparate techniques, rather superficial but nevertheless useful as a glossary. After this comes a chapter in epidemiological methods including issues of case definition, measures of psychiatric morbidity, sampling methods and other important epidemiological concepts which are applicable to all clinical research.

A chapter on single patient research designs is very welcome. These are difficult to interpret but more within the scope of individual trainees. With this chapter in hand many trainees might be encouraged to carry out intensive research on one or two cases.

Two chapters on psychotherapy and psychological treatment will also be helpful. This is an area which many research councils find difficult to fund because of the paucity of scientifically-valid protocols sent to them. Next comes a chapter on psychometrics dealing with issues of validity and reliability. It is called "Principles of psychological assessment" but it might just as well be called "Principles of psychiatric measurement". There are three chapters on rating instruments, one for general psychiatry, one for psychotherapy and one for child psychiatry and eating disorders. Together with my own book *The Instruments of Psychiatric Research,* these provide any budding researcher with access to a large number of instruments and a way into the literature. Finally, two chapters on writing up and using computers are very practical and helpful.

It is to be welcomed that at last the area of research methods in psychiatry is receiving more prominence. There have been books before, notably a Maudsley monograph on the subject, but they are now out of date This one is likely to be the market leader. However, readers should not ignore another recent volume by Parry & Watts, *Behavioural and Mental Health Research: A Handbook of Skills and Methods.* That would be a good complimentary book to the one under review here.

CHRIS THOMPSON, *Professor of Psychiatry, University of Southampton*

Families, Services and Confusion in Old Age. By ENID LEVIN, IAN SINCLAIR and PETER GORBACH. Aldershot: Gower Publishing. 1989. 328 pp. £29.50.

This book is based on a project carried out by the research unit of the National Institute for Social Work. It studies the problems faced by supporting relatives of confused elderly people, and looks at how such relatives feel about these and how they handle them. It also evaluates the effects of a range of standard community services and of residential care upon supporting relatives.

In the introductory chapter the authors highlight the growing interest in the needs of supporters of the demented elderly brought about by a policy of community care They refer to studies on families supporting demented elderly relatives and to those that have looked at the demands the demented elderly make on services.

The rest of the book divides broadly into three sections. The first section carries out a detailed survey of 150 families and examines the practical, behavioural, interpersonal and social problems that the supporting relatives faced. The second section looks at the consequences of providing support. The third section describes the forms of help potentially available to the supporters, discusses its allocation and evaluates its effects.

The concluding chapter is probably of most interest to all service providers. In it, the authors draw together the evidence from the study and strengthen the case already made by others for 'supporting the supporters'. They also comment on issues relating to training, resources, planning and co-ordination of support services. In doing this the authors argue the case for more joint action between health and social services.

Although an attempt has been made to set this book out as clearly as possible, it is not an easy book to read. The print is unnecessarily poor. While bar charts and tables throughout help, the amount of data in the text and the style in which it is presented make the information difficult to assimilate.

Nevertheless, this book will be a useful addition to any hospital library for the use of those working with the elderly and those with a research interest in the field.

SUE WEST, *Lecturer in Mental Health, University of Bristol*

Assessment of Addictive Behaviour. By D. M DONOVAN and A. MARLATT. London: Century Hutchison. 1988. 497 pp. £30 00

The late Queen Mary is reported to have said of Sir Alfred Bossom "Silly name really, neither the one nor the other". This multiple-authored book suffers from the same problem of a very unsatisfactory title. The authors are psychologists involved with counselling and behavioural modification techniques for a wide range of problems They have tried to categorise all these (including obesity and smoking cannabis) as 'addictions' by using a definition that is unduly broad and over-inclusive. This has caused problems for some authors. For example, the chapter on behavioural and cognitive behavioural assessment of obesity does not mention addiction or addictive behaviour.

The definition they use is that "... addiction is a complex progressive behaviour pattern having biological, sociological and behavioural components. What sets this behaviour pattern apart from others is the individual's overwhelmingly pathological involvement in or attachment to it, and reduced ability to exert personal control over it". This is unsatisfactory since it appears to include any repetitive behaviour such as an obsessional neurosis (e.g. compulsive handwashing), masturbation or regularly exceeding the speed limit. From the vantage point of the Parole Board there are many young tearaways who have an overwhelmingly pathological involvement in and attachment to 'taking (cars) without the owners consent' (TWOC). They have a subjective compulsion to continue and a reduced ability to exert personal control over this, as is shown by continuation despite fines, disqualification and imprisonment. Other behavioural modification techniques than those used presently by the law might help this group, but to describe car theft as an 'addiction' would be absurd

As with many multi-authored books the quality varies markedly. Some parts are helpful, some are not, and the problems of overlapping and repetition have not been solved. The intended audience for the book includes "students, clinicians (therapists and counsellors) and researchers .. who are provided with an update and expansion of previously available information on the assessment of addictive behaviours". It brings together a wide range of information on how to assess a group of problems, including those with alcohol, tobacco, dependence-producing drugs, obesity, eating disorders and smoking cannabis.

Dr Samuel Johnson, who must have been an early master of the rapid eyeball-scan approach to literature "had a peculiar facility in seizing at once what was valuable in any book, without submitting to the labour of perusing it from beginning to end". A trainee counsellor wondering how best to assess and help a client with problems that might respond to behavioural counselling, could make use of Johnson's technique to extract the relevant information from this book. Thirty minutes should suffice. Reading the book from cover to cover would be a mistake.

Thomas Bewley, *Consultant Senior Lecturer,*
St George's Hospital Medical School, London

Four Therapeutic Approaches to the Borderline Patient: Principles and Techniques of the Basic Dynamic Stances. By Andrew Druck. New Jersey: Jason Aronson. 1989. 440 pp. $40.00.

Druck is a New York psychoanalyst and teacher of psychoanalysis. His book addresses the treatment problems raised by borderline patients by looking at four different theoretical and technical approaches to the analysis of them.

The four models Druck examines are: Kernberg's model, the representational deficit/self deficit models, the ego deficit model and a model he calls 'modifications of classical technique'. In the first part of his book the author outlines the theoretical bases of these different models, the underlying assumptions and the implications, therefore, for technique A whole early chapter is given over to a case presentation. Here, a number of sessions are reported together with demonstrations of how the material offered might be used by various practitioners. This is interesting, but perhaps mainly because one ends up with a great deal of sympathy for the therapist whose struggles with her difficult patient are so vividly recorded and thoroughly examined.

Throughout the book many authors are quoted in detail, almost all of them American. This is very evident in a chapter which explores the distinctions between supportive and expressive therapies, and in the section entitled "Variations in the therapist's stance". These debates raise many interesting points, but for readers who are not very familiar with the American literature they can become confusing, as the authorities quoted quote each other in a seemingly endless regression. There are thirteen pages of references at the end of the book; Druck has plainly engaged in a scholarly attempt to grapple with the profusion of the literature.

One of the most important figures in this field in the USA is, of course, Otto Kernberg. Druck devotes a quarter of his book to "A critical inquiry into Kernberg's technique". He gives a clear account of Kernberg's views and rigorously tackles those he disagrees with, particularly where he feels that Kernberg's emphasis ignores important issues like the fostering of a working alliance or the transference implications of the analyst arranging for the patient's care outside sessions, by, for example, admission to hospital.

Druck acknowledges that "my bias is toward the model I have termed 'modifications of classical technique' " (p. 55), and as the book proceeds this becomes increasingly apparent. The last section is entitled "Expansion possibilities within classical technique" and here, interestingly, he begins to quote some British authors. He addresses changing conceptions of the therapist's role and discusses the left and right wings of classical technique. Important themes like the rule of abstinence are well aired – he has earlier explored the famous example of Freud giving a meal to his patient the Rat Man. Patrick Casement is quoted at some length on the issue of gratification, and Michael Balint on regression. The overall focus becomes that of what adjustments may be made to enable the therapy of borderline patients to proceed while keeping the treatment psychoanalytic. This is what really interests the author, and he has set about exploring it by way of comparing different models. In particular, I think he has been debating whether a deficit model of the psychopathology is more therapeutically useful than a conflict one, and whether the two are, in fact, mutually exclusive.

It will be seen that this book is quite profoundly clinical. It is fairly hard work to read, although the reader is rewarded by the ideas that are constantly sparked off. There are many pertinent case illustrations; altogether it is rather like being involved in a series of lively seminars, and I suspect that Druck would probably be a good teacher.

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Human Sexuality and Its Problems. (2nd edn) By John Bancroft. Edinburgh: Churchill Livingstone. 1989. 745 pp. £24.95.

Recent advances in our understanding and management of erectile problems, the AIDS epidemic, and the 'apparent' increase in sexual abuse of children, have dramatically changed sexual attitudes and practices. This book was re-written and doubled in size to reflect such changes. As a one-author book, it has advantages over multi-author texts in that it gives a clear message, is consistent and maintains a sense of direction and unity. However, such a book is inevitably deficient in some areas and detailed in others, depending on the author's areas of expertise, and personal choices. Bancroft is fully aware of this and even indicates some of the deficiencies himself. He does not pretend that he can give a comprehensive cover for such a vast subject.

American textbooks on human sexuality dominated the scene for a long time and tended to cite American research and scarcely mention British or other European works. This book, however, redresses the balance, by giving equal opportunity for British and other European works to contribute to the debate, although it is still based on many US works. The author's own work features in all chapters but is critically discussed in the same impartial way.

Most books which discuss the anatomy, physiology and pathology of the penis and vagina in such a depth, tend to pay very little attention to the human being to whom these organs belong and even less attention to the social network, the culture and the attitudes and prejudices of the professional who studies or treats them. This colossal work covers adequately the basic science and the medical and clinical aspects of human sexuality as well as its psychological, social, political and moral aspects. The author challenges many of the dogmatic views on human sexuality, criticises the evidence for them and puts forward new opinions supported by evidence from recent research. He gives clear and forthright views on emotive subjects like homosexuality, pre-marital and extra-marital sex, child sexual abuse, feminism, rape victims and even on child sexuality. With hypocrisy and double standards still abounding in the area of sexuality, to take on such a task you have to either be very brave or an expert on the subject. John Bancroft is both.

No-one can write so extensively about human sexuality without their personal prejudices and moral attitudes shaping the discussion. However, Bancroft explicitly mentions what his personal opinions are and thus takes full responsibility for them. You can disagree with some of these views, you can even attack him for them, but you can't help admiring him for his honesty and courage.

There are two minor irritations although they do not really devalue the book in any way. Firstly, a number of spelling mistakes exist which the publisher should have corrected. Secondly, the references are listed at the end of every chapter so there is no author's index to help readers to follow the work of one particular author through the various chapters.

No medical or psychiatric library will be complete without a copy of this book. No clinician or research worker in the area of human sexuality can afford not to have a personal copy to dip into whenever there is a clinical or research problem. The reader will never be disappointed as there will always be a useful, thought-provoking idea and a wealth of references for further reading.

Even at the price of £25 a copy, this book is excellent value for money.

WAGUIH R. GUIRGUIS, *Psychosexual Clinic,*
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Between Freud and Klein: The Psychoanalytic Quest for Knowledge and Truth. By ADAM LIMENTANI. 1989. 281 pp. £30.00.

This book is a collection of papers which have all been previously published elsewhere, ranging widely over the last 25 years. Apart from the first, they are arranged chronologically, about half are centred on the author's work with the perversions at the Portman Clinic while the others cover topics such as acting out and the negative therapeutic reaction

The book, which takes its title from a paper originally published in Italian, is subtitled "The psychoanalytic quest for knowledge and truth" and it seems to me that the author sustains his quest for these elusive goals with admirable honesty and at times with what amounts to painful soul-searching; this was perhaps most evident in the discussion on training analyses in chapter five. However, it was in the more clinical chapters where the author's eloquent prose style came particularly into its own; the case histories were a delight to read

In the chapter on the negative therapeutic reaction Limentani states (p. 156) "the essential element of a negative therapeutic reaction is that the analytic work which has lovingly and meticulously been carried out is laid in fragments before us". Such theoretical ideas in the collection were clearly and succinctly expounded, and the developments from Freud's original ideas are carefully traced. The only chapters where the expositions were not clear were the contiguous papers entitled "On some aspects of human violence" and "On the psychodynamics of drug dependence". In these there seemed at times an uneasy relationship between the analytic and the sociological aspects of the problems, with the risk at times of generalisation, although the author tried to steer clear of this.

I suspect I am cavilling at these papers because of their contrast with the beautifully described case histories throughout the book, for example, the description of 'Jenny' entitled "A 20-year study of childhood delinquency evolving into adult neurosis". This book is written by an analyst whose work should be of interest to all those involved with the internal world, and the author manifestly demonstrates that his position "between Freud and Klein" is not constricted but indeed a creative one.

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Development and Psychopathology: Studies in Psychoanalytic Psychiatry. By CLIFFORD YORKE, STANLEY WISEBERG and THOMAS FREEMAN. New Haven and New York: Yale University Press. 1989. 243 pp. £22.50/$36.00 (hb).

This book is intended for those psychiatrists who have a serious interest in gaining further intellectual acquaintance with the psychoanalytic theory of cases similar to those they see in everyday practice. It contains chapters on anxiety states, neuroses, character neuroses and disorders, and the common major psychoses.

The authors are of the firm viewpoint that there is no proper psychiatry "without a coherent and comprehensive psychology, and that the developmental perspective of psychoanalysis is indispensable for the *psychological* understanding of psychiatric states".

It is not an account of psychoanalytic practice or therapy nor does it directly discuss management, but it is intended to make a major contribution to the clinician's understanding of the powerful internal psychological forces that his or her patients are contending with and of why a particular patient may become disturbed in a particular way at a particular time.

The specific psychoanalytic framework is very much classic Freudian with the addition of some important developmental concepts elaborated by Anna Freud and her colleagues (the authors are all eminent participants in the research work of the Anna Freud Centre, in London, as well as having extensive experience of National Health Service psychiatry).

Readers will need to be already acquainted with basic psychoanalytic theories of psychosexual development and have a working knowledge of the structural model of the mind and the fundamentals and terminology of classical Freudian metapsychology. Those so equipped will find a wealth of evidence to show how much Freud's basic hypotheses continue to have extraordinary explanatory value. They will also find that the authors' familiarity with child development brings alive the earlier difficulties – "developmental disharmonies, deviations, fixations and arrests" – and helps make sense of why their cases break down at later points in their lives.

It may surprise many how much interest in classification is expressed by the authors, yet how useful it is to read, for example, of the different clinical picture and metapsychology between manic disorders when there has been loss and those when there has been frustration. It is also refreshing to get both a clear clinical and theoretical account of the differences between symptom neuroses, character neuroses and non-neurotic character disorders. (However, I have yet to grasp the differentiation that they make between character and personality).

Readers will moreover learn a lot from the careful description of the pre-psychotic states of documented cases and of the relationship between neurotic states and psychotic ones. They will be treated to an excellent description of different theoretical viewpoints that stem from the difficulty in determining what are primary and what secondary psychological disturbances in the schizophrenic states. They will also learn about the important differences in opinion about the state of the ego, i.e. whether the relevant part of the ego becomes de-differentiated from the other agencies or whether it functions in a psychotic or infantile form in schizophrenic disorders.

This is a book of considerable scholarship and erudition. To reach the wide psychiatric audience that it is intended for, it would be helpful if there were at least a couple of chapters at the beginning, giving a basic introduction, in simpler language, of the key developmental and metapsychology concepts that are used throughout.

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SILVERSTONE, T. & GOODALL, E. (1984) The clinical pharmacology of appetite suppressant drugs. International Journal of Obesity, 8 (suppl. 1), 23–33.

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——, —— & GOODALL, E. (1988) The prevalence of obesity in patients receiving depot antipsychotics British Journal of Psychiatry, 153, 214–217.

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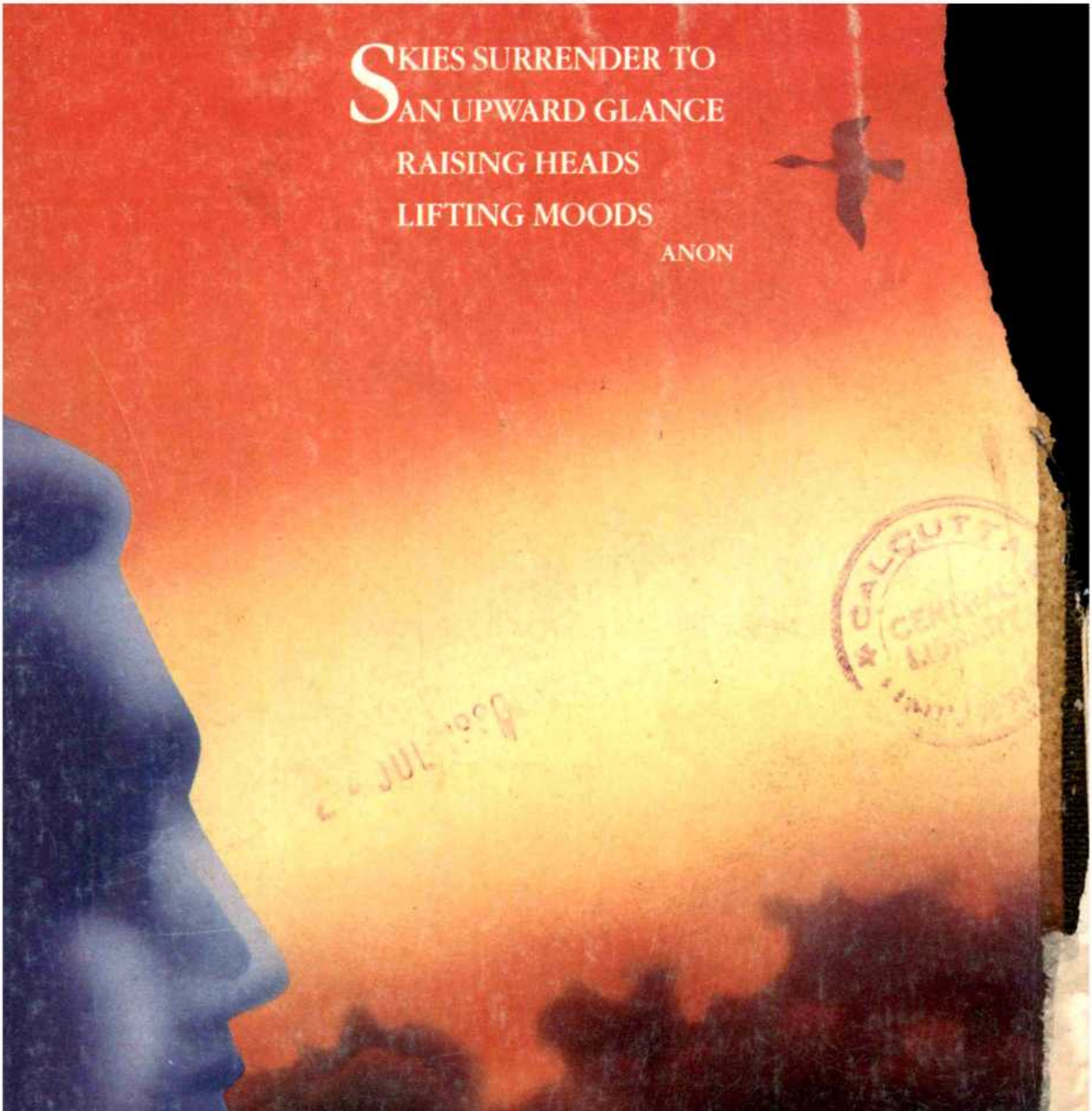

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